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16. ABSTRACT

A review of the 1980 world literature was conducted on the effects of pollution on freshwater fish. This review contained 401 references and included papers on water quality such as dissolved gases and pH; chemical pollutants such as insecticides, herbicides, piscicides and anesthetics; inorganics, organics and industrial and municipal effluents including chlorine and wastewater and pulp and paper. Papers from reviews and symposia and those dealing with physical factors were also included. Within the above sections, papers were organized into general articles, surveys, toxicology, bioaccumulation, physiology and behavior.

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Effects of pollution on freshwater fish

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REVIEWS AND SYMPOSIA

The current status of aquatic toxicology, including various endpoints, fish species used, and methodologies, was reviewed by Tucker and Leitzke.[1] Methodological variations caused larger differences in results than the endpoints chosen, and there was no evidence for a "most sensitive" species. Extrapolations among species were poorly developed and were generally unnecessary, because important species can be directly tested. Spehar *et al.*[2] reviewed 478 references concerning the effects of pollution on freshwater fish. Subject matter included water quality, physical and chemical pollutants, and domestic and industrial wastes. Perkins[3] reviewed the need for sublethal toxicity studies by describing various types of sublethal tests, test chemistry needs, selection of organisms, and problems of data applicability. The author concluded that exposures that are debilitating but not lethal will prove to be of most practical value. Two symposia were jointly sponsored by the U. S. Environmental Protection Agency[4] and the USSR on the effects of pollutants upon aquatic ecosystems. The subjects ranged from general limnology to specific cases of fish toxicity. Effects of insecticides, cyanides, and ammonia on fish were described. BIOKON[5] compiled and reviewed information on sources, fate, and aquatic toxicity of 1 500 chemicals used in the member states of the Commission of European Communities. Some of the types of chemicals reviewed were organophosphorus compounds, metals, persistent synthetic solids, and petroleum oils and hydrocarbons. The literature was reviewed on the toxicity of components of petrochemical effluents to aquatic life.[6] Toxic concentrations of many of the components were lower than existing standards, and dilution was necessary even on some treated wastes. Buikema *et al.*[7] reviewed the significance of phenolics in aquatic ecosystems. Tables of acute toxicity to fish were given, and various effects of phenolics on fish were included. In addition, population and community effects were discussed, as well as fate and transport of these compounds. The U. S. Department of Commerce[8] updated a previous bibliography on polychlorinated biphenyls (PCBs) in the environment and added 74 new references. There were a number of abstracts on effects on freshwater fishes. The effects of hexachlorobenzene on aquatic organisms, including freshwater fishes, were summarized by Craig.[9] The data were presented entirely in tabular form and included toxicity and residue formation. A comprehensive review of the health and environmental effects of 1,2-dichloroethane revealed very little data on toxicity to fishes.[10] For the fish species tested, acute toxicity appeared relatively low, with the LC_{50} ranging from 100 to 1 000 mg/l.

Reviews of the literature on the toxicity of pulp and paper mill effluent to aquatic organisms were made by Hutchins[11] and Poole *et al.*[12] The toxicity of different process effluents and the use of fish bioassays to measure toxicity, as well as avoidance and population impacts, were discussed. Leland *et al.*[13] and Phillips and Russo[14] reviewed the literature on bioaccumulation and toxicity of heavy metals to fishes. The authors discussed the importance of speciation of metals as it affects toxicity and the significance of residues to other consumers of various metals. In addition, field residue data were emphasized. The uses of Hg in the industry and agriculture of Thailand and its effects as a toxic pollutant (causing fish kills) were reviewed by Lohani and Muttamara.[15] The present state of Hg problems and control measures were also discussed. A complete review of the toxicity of Cu to aquatic life was published by Taylor.[16] A graphic presentation and extensive bibliography on Cu were included. A review of the use of potassium permanganate in fisheries[17] included extensive data on its acute effects to several fish species. The data were compiled in tabular form, along with a brief summary of

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the factors affecting toxicity. A symposium covering the effects of surface mining on fish and wildlife was held in December 1978 in Morgantown, W. Va.[18] Five papers were included that dealt with the effects on freshwater fishes of mine operation and on methods for measuring these effects.

The literature on the effects of stream sediment on aquatic organisms, including fish, was summarized.[19] Effects such as suffocation were found in eggs and young fish, especially salmonids. Several methods for measuring sediment in streams were described, but most were labor intensive. McLeod[20] reviewed the development of gas bubble disease in fish exposed to supersaturated water, adaptation to supersaturation, and supersaturation in the environment. Also discussed was the response of fish in general, and the Atlantic menhaden in particular, to supersaturated water.

WATER QUALITY

Dissolved gases. The effects of an artificial O_2 supply on fish production in ponds were examined by Satomi *et al.*[21] Using an O_2 generator and a mixing device, the authors found that increased O_2 supply in the ponds is very efficient in achieving higher production of carp and tilapia despite higher concentrations in nitrites in the water. However, Fast[22] cautioned against artificial aeration of lakes with bottom releases, since gas supersaturation can occur if the bottom waters are released for fish culture or for tail-water replenishment. Measurements of nitrogen and argon gases in an artificially aerated lake showed concentrations ranging from 105% saturation at the surface to 140% at the bottom. DeInfante *et al.*[23] attributed a massive fish and zooplankton kill in Lake Valencia, Venezuela, to a high hydrogen sulfide concentration and O_2 depletion. This was caused by anoxic conditions from the accumulation of large amounts of substances that were mixed throughout the water column by suddenly increased winds.

Of the sockeye salmon (*Oncorhynchus nérka*) smolts held by Nebeker *et al.*[24] for 3 weeks in 117% air-supersaturated freshwater, 70% died; at 115%, 5% died. However, supersaturation did not cause any residual effects on smolt survival in sea water. When the severely stressed survivors from these concentrations and others held in 113, 110, 108, and 100% saturated water were transferred to sea water, they recovered from gas-bubble disease, and none died during the 10-day holding period. The effects of different temperatures on survival of juvenile steelhead trout (*Salmo gairdneri*), juvenile chinook (*Oncorhynchus tshawytscha*), coho salmon (*Oncorhynchus kisutch*), and sockeye salmon at constant air-supersaturation concentrations were also determined.[25] Increased temperature in the 8°–20°C range caused a significant increase in steelhead and chinook mortality, but no significant effect on coho or sockeye mortality in supersaturated water concentrations between 115 and 120%. The 3-day LC_{50} for salmon (*Salmo salar*) smolts exposed suddenly to low constant concentrations of dissolved oxygen (DO) was 3 mg/l in fresh water and about 2.5 mg/l in 30 and 80% sea water.[26] The same LC_{50} values were obtained even when the fish were acclimated for several days to low concentrations of DO and high salinity. From both laboratory and field studies, Casselman[27] related the effects of major environmental factors such as temperature, photoperiod, and DO on growth, survival, activity, and exploitation of northern pike (*Esox lucius*). It was shown that the lower incipient lethal DO concentration is directly related to temperature for this species and that the northern pike, being extremely tolerant of low DO in shallow lakes, can survive minimum DO concentrations of 0.3 mg/l.

Rosenkranz *et al.*[28] reported that the 96-hour LC_{50} for adult white perch (*Morone americana*) exposed to ozone-produced oxidants (OPO) is 0.22 mg/l. Sublethal response and recovery studies showed significant decreases in blood pH and hematocrit increases over the 96-hour test period in fish exposed to 0.10–0.15 mg OPO/l. Histological changes occurred in the gill within a 24-hour exposure at all concentrations tested (0.01–0.15 mg OPO/l). However, gill repair was evident after 14 days of recovery. In another study, Richardson *et al.*[29] exposed 12-hour old striped bass (*Morone saxatilis*) eggs over the concentration range of 0.005–0.10 mg OPO/l and evaluated the effects on egg development at 24 and 42 hours after fertilization as well as the effects on two prolarval stages (24- and 48-hour posthatch). Exposures in both fresh and estuarine water showed that OPO is more toxic in freshwater, that eggs are more resistant than prolarvae, that delayed hatching occurs in eggs exposed to 0.05 and 0.10 mg/l in estuarine water, and that delayed hatch organisms survive better than hatched larvae at the same developmental stage. The acute

toxicity curve for dissolved ozone (O_3) yielded a 96-hour LC_{50} value of 9.3 μg O_3/l for juvenile rainbow trout (*Salmo gairdneri*).[30] Death in acute exposure was most likely caused by severe gill lamellar epithelial tissue destruction accompanied by massive hydromineral imbalances. Because partial chronic tests showed little biological damage at 2 μg/l and some histopathological changes in gill tissue at 5 μg/l, it was suggested that 2 μg/l is the maximum safe exposure level for salmonids, pending completion of life cycle testing and development of ozone-specific analytical methods. Paller and Heidinger[31] assessed the effects of water containing ozone bluegill sunfish (*Lepomis macrochirus*) and concluded that ozone is highly toxic to bluegills, with safe limits for prolonged exposure probably lying well below 0.01 mg/l. The 24-hour LC_{50}, as determined by standard bioassay, was 0.06 mg/l, while the LC_{50} was 0.32 mg/l for periodic dosing consisting of six 30-minute exposures 8 hours apart. Bluegills held in concentrations between 0.01 and 0.02 mg/l showed irregular respiration, decreased activity, cessation of feeding, and random mortality during the 6-week exposure.

Hillaby and Randall[32] tested acute ammonia toxicity in rainbow trout by intra-arterial injection of NH_4Cl and NH_4HCO_3. The same dose of each was required to kill fish even though NH_4Cl decreased arterial pH while NH_4HCO_3 increased arterial blood pH. Measured ammonia levels in the blood provided evidence for ammonia excretion at the gills in the un-ionized form. Twenty-four-hour LC_{50} values for channel catfish (*Ictalurus punctatus*) fingerlings exposed to total ammonia at pH 7, 8, and 9 were 263.6, 38.8, and 4.5 mg NH_3-N/l, respectively.[33] At pH 7, water enrichment to 200 mg/l Ca significantly increased the 24-hour LC_{50} to 256.3 mg/l. There were no differences in hematocrit, percentage of total plasma protein, or plasma and muscle chloride between controls and those fish exposed to 24 mg NH_3-N/l for 12 hours at pH 7 and 8. However, plasma sodium decreased significantly at pH 8. The 72-hour LC_{50} for *Tilapia aurea* exposed to un-ionized ammonia was 2.35 mg N/l.[34] When the fish were acclimated at sublethal concentrations between 0.43 and 0.53 mg un-ionized ammonia/l for 35 days, no mortalities occurred within 48 hours after exposure to concentrations as high as 3.4 mg/l. In both cases, however, histopathological changes occurred in the gills. In a 31-day growth trial conducted by Colt,[35, 36] the growth of channel catfish was reduced linearly when exposed to ammonia (as NH_3-N) concentrations between 48 and 989 μg/l. A 50% reduction in growth occurred at 517 μg/l, and no growth occurred at 967 μg/l. Above 989 μg NH_3-N/l, mortality increased significantly, resulting in a 96-hour LC_{50} of 1 600 μg/l.

Baird *et al.*[37] investigated anomalous fish mortality in static bioassays of treatment plant effluents containing ammonia and recommended that low-flow O_2 aeration be used to control unrepresentative fish kills. These occurred because of a lethal shift in the NH_4^+-NH_3 equilibrium caused by CO_2 stripping when ordinary aeration was used and a subsequent rise in pH from 7.2 to 8.0 and above. During the course of these investigations, the 96-hour LC_{50} for golden shiner (*Notemigonus crysoleucas*) exposed to ammonia was assessed as 0.99 mg NH_3-N/l. Griffith *et al.*[38] found that killifish (*Fundulus heteroclitus*) held in fresh water showed higher mortality than those in sea water or 1/3 sea water when exposed to a constant concentration of 0.4 mg urea/l. Freshwater fish ceased feeding sooner and had lower control plasma sodium levels than seawater-exposed fish.

The results of a 5-year study that determined the acute and chronic toxicity of HCN to seven fish species and two invertebrates were reported by Smith *et al.*[39] Chronic tests showed that, on the basis of spawning success, the maximum acceptable toxicant concentration (MATC) for brook trout (*Salvelinus fontinalis*) was between 5.7 and 11.2 μg HCN/l and that the concentration having no adverse effect on egg production in fathead minnows (*Pimephales promelas*) was between 12.9 and 19.6 μg HCN/l. For bluegills, no spawning occurred in long-term tests at concentrations as low as 5.2 μg HCN/l. Another study[40] on HCN empirically examined the validity of various models for predicting joint toxicity to fish, based on lethal and sublethal responses to individual toxic components in the mixtures. In acute tests (Table I) with fathead minnows and rainbow trout, log-dosage mortality curves showed that Zn-HCN and ammonia-HCN mixtures were more toxic and that Cr-HCN was less toxic than predicted by the strictly additive toxic unit and additive index approach. Sublethal joint action of individual toxicants was not predictable from existing models, and it was concluded that a need still exists for development of a valid

TABLE I. Summary of the acute and chronic toxicity of inorganic and organic pollutants to freshwater fish.

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Acetone[202]	Goldfish (*Carassius auratus*)	24-hour LC50, >5 000 mg/l	S[a]	20	pH 6–8
Acrolein[202]	Goldfish	24-hour LC50, <0.08 mg/l	S	20	pH 6–8
Acrylamide[202]	Goldfish	96-hour LC50, 160 mg/l	S	20	pH 6–8
Adogen 283[207]	Rainbow trout (*Salmo gairdneri*) Fry	120-hour LC50, 1.3–9.7 mg/l	S	10–20	pH 7.0 HD[b] 180
Alamine 336[207]	Rainbow trout Fry	120-hour LC50, 0.9–2.5 mg/l	S	10–20	pH 7.0 HD 180
Aldrin (30% EC)[102]	Khard (*Colisa fasciatus*)	96-hour LC50, 0.021–0.10 mg/l	S	17–32	pH 6.4–8.0
	Patra (*Notopterus notopterus*)	96-hour LC50, 0.49–4.6 µg/l	S	17–32	HD 60–108 pH 6.4–8.0 HD 60–180
Aliquat 336[207]	Rainbow trout Fry	120-hour LC50, 0.5–0.87 mg/l	S	10–20	pH 7.0 HD 180
Alkyl ethoxylate[236]	Fathead minnow (*Pimephales promelas*)	96-hour LC50, 1.2–1.4 mg/l	S	15	pH 7.4–7.8 HD 100–250
Allyl alcohol[202]	Goldfish	24-hour LC50, 1 mg/l	S	60	pH 6–8
Allyl bromide[202]	Goldfish	24-hour LC50, <0.8 mg/l	S	20	pH 6–8
Allyl chloride[202]	Goldfish	24-hour LC50, 10 mg/l	S	20	pH 6–8
Allyl glycidyl ether[202]	Goldfish	96-hour LC50, 30 mg/l	S	20	pH 6–8
Amberlite LA-1 (secondary amine)[207]	Rainbow trout Fry	120-hour LC50, 1.2–6.4 mg/l	S	10–20	pH 7.0 HD 180
Amitrol-T[150]	Mosquitofish (*Gambusia affinis*)	96-hour LC50, 2 100 mg/l	S	21–22	—
Ammonia[40]	Rainbow trout Juvenile	96-hour LC50, 0.697 mg/l	F[c]	10	pH 8.0 HD 220
Ammonia (NH_3-N)[40]	Rainbow trout Juvenile	96-hour LC50, 35.7 mg/l	F	10	pH[d] 8.0 HD 220
Ammonia (NH_3-N)[20]	Channel catfish (*Ictalurus punctatus*) Fingerling	24-hour LC50, 263.6 mg/l	—	21–24	pH 7 HD 40
	Fingerling	24-hour LC50, 38.8 mg/l	—	21–24	pH 8 HD 40
	Fingerling	24-hour LC50, 4.5 mg/l	—	21–24	pH 9 HD 40
Ammonia (NH_3-N)[36]	Channel catfish Juvenile	96-hour LC50, 1 600 µg/l	F	28	pH 8.3
Ammonia (NH_3-N)[37]	Golden shiner (*Notemigonus crysoleucas*)	96-hour LC50, 0.99 mg/l	S	—	pH 8.3
Ammonia (un-ionized)[24]	*Tilapia aurea*	72-hour LC50, 2.35 mg/l	F	24.5–28.0	pH 7.3
Ammonium fluoride[224]	Fathead minnow Juvenile	96-hour LC50, 363.8 mg/l	S	22	pH 7.2–7.9 HD 40–48
Aresin[160]	*Sarotherodon mossambicus*	96-hour LC50, 54 mg/l	S	28.5	pH 8.5
	Channa punctatus	96-hour LC50, 104.4 mg/l	S	28.5	
Arsenic (mono sodium methyl arsonate)[150]	Mosquitofish	96-hour LC50, 1 300 mg/l	S	21–22	—
	Mosquitofish	96-hour LC50, 182 mg/l	S	21–22	—

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Arsenic (sodium arsenite)[238]	Muskellunge (*Esox masquinongy*)				
	Fry	5-day LC50, 0.05 mg/l	S	15	pH 7.2–7.7
Arsenic (sodium arsenite)[200]	Muskellunge				
	Newly hatched fry	96-hour LC50, >5 mg/l	S	15	pH 7.2–7.7
	Swim-up fry to 5-week-old fry	96-hour LC50, 1.1–16 mg/l	S	15	
Arsenic (sodium arsenite)[139]	Mosquitofish	96-hour LC50, 59 mg/l	S	21–22	—
Arsenic trisulfide[254]	Fathead minnow				
	Juvenile	96-hour LC50, 135.1 mg/l	S	22	pH 7.2–7.9 HD 40–48
Barite (barium sulfate)[280]	Rainbow trout				
	Fry	96-hour LC50, 76 000 mg/l	S	10.5	pH 8.2 HD 340
Ben-Ex (polyvinyl acetate, maleic anhydride)[280]	Rainbow trout				
	Fry	96-hour LC50, 660 mg/l	S	10.5	pH 8.2 HD 340
Bentonite (montmorillonite clay)[280]	Rainbow trout				
	Fry	96-hour LC50, 19 000 mg/l	S	10.5	pH 8.2 HD 340
Benzene[281]	Dolly varden (*Salvelinus malma*)				
	Juvenile	96-hour LC50, 12.0 µg/l	S	8	—
	Smolt	96-hour LC50, 11.9 µg/l	S	8	—
	Chinook salmon (*Oncorhynchus tshawytscha*)				
	Juvenile	96-hour LC50, 11.7 µg/l	S	6–9	—
	Coho salmon (*Oncorhynchus kisutch*)				
	Juvenile	96-hour LC50, 14.1 µg/l	S	8–9	—
	Embryo	96-hour LC50, 542 µg/l	S	8–9	—
	Alevin	96-hour LC50, ~50 µg/l	S	8–9	—
	Emergent fry	96-hour LC50, 9.8 µg/l	S	8–9	—
	Sockeye salmon (*Oncorhynchus nerka*)				
	Smolt	96-hour LC50, 10.8 µg/l	S	6	—
	Arctic grayling (*Thymallus arcticus*)				
	Juvenile	96-hour LC50, 14.7 µg/l	S	9	—
	Slimy sculpin (*Cottus cognatus*)	96-hour LC50, 15.4 µg/l	S	9	—
	Threespine stickleback (*Gasterosteus aculeatus*)	96-hour LC50, 24.8 µg/l	S	5–8	—
	Pink salmon (*Oncorhynchus gorbuscha*)				
	Embryo	96-hour LC50, 339 µg/l	S	4	—
	Alevin	96-hour LC50, ~50 µg/l	S	4	—
	Emergent fry	96-hour LC50, 5.3 µg/l	S	4	—
	Outmigrant fry	96-hour LC50, 17.1 µg/l	S	4	—
Benzene[282]	Goldfish	24-hour LC50, 46 mg/l	S	20	pH 6–8
Benzo(a)pyrene[339]	Rainbow trout	10-day BCF,[4] 920	F	13	pH 7.0 HD 45
Benzoyl chloride[254]	Fathead minnow				
	Juvenile	96-hour LC50, 34.7 mg/l	S	22	pH 7.2–7.9 HD 40–48
Benzyl chloride[254]	Fathead minnow				
	Juvenile	96-hour LC50, 4.7–7.8 mg/l	S	22	pH 7.2–7.9 HD 40–48

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
B-Free (surfactant)[200]	Rainbow trout				
	Fry	96-hour LC50, 18 mg/l	S	10.5	pH 8.2 HD 340
Bicyclo(2.2.1)hepta-2,5-diene[202]	Goldfish	24-hour LC50, 46 mg/l	S	20	pH 6–8
Bladex[150]	*Sarotherodon mossambicus*	96-hour LC50, 11.3 mg/l	S	28.5	pH 8.5
	Cirrhinus mrigala	96-hour LC50, 6.3 mg/l	S	28.5	
5-Bromoindole[201]	Fathead minnow	32-day BCF, <10	F	25	—
5-Bromoindole[200]	Fathead minnow	32-day BCF, 14	F	25	pH 7.5 HD 45.5
o-Bromonitrobenzene[202]	Goldfish	24-hour LC50, 46 mg/l	S	20	pH 6–8
i-Butyl alcohol[202]	Goldfish	24-hour LC50, 2 600 mg/l	S	20	pH 6–8
n-Butyl alcohol[202]	Goldfish	24-hour LC50, 1 900 mg/l	S	20	pH 6–8
sec-Butyl alcohol[202]	Goldfish	24-hour LC50, 4 300 mg/l	S	20	pH 6–8
N-n-Butylimidazole[202]	Goldfish	96-hour LC50, 30 mg/l	S	20	pH 6–8
Cadmium[221]	Steelhead trout (*Salmo gairdneri*)	408-hour LC50, 5.2 μg/l	F	9.6	pH 7.5 HD 54
	Coho salmon				
	Adult male	215-hour LC50, 3.7 μg/l	F	10	pH 7.3 HD 22
Cadmium[204]	Sockeye salmon				
	Newly hatched fry	168-hour LC50, 4 500 μg/l	F	5–8	pH 7.6
	First feeding fry	168-hour LC50, 1 000 μg/l	F	5–8	HD 83
	Fry	168-hour LC50, 8 μg/l	F	5–8	
	Smolt	168-hour LC50, 360 μg/l	F	5–8	
	Pink salmon				
	Newly hatched fry	168-hour LC60, 3 200–3 600 μg/l	F	5–8	
	First feeding fry	168-hour LC50, 2 700 μg/l	F	5–8	
Cadmium[203]	Goldfish	48-hour LC50, 5–10 mg/l	S	10–12	—
Capacitor 21[207]	Channel catfish				
	Embryo-larvae	4-day LC50, 5.2 μg/l	R	29–31	pH 7.6–8.1 HD 90–115
	Goldfish				
	Embryo-larvae	4-day LC50, 14.9 μg/l	R	20–24	
	Rainbow trout				
	Embryo-larvae	4-day LC50, 1.6 μg/l	R	13.5–14.3	
	Redear sunfish (*Lepomis microlophus*)				
	Embryo-larvae	4-day LC50, 6.5 μg/l	R	20–24	pH 7.6–8.1 HD 90–115
	Embryo-larvae	4-day LC50, 13.5 μg/l	F	20–24	pH 7.6–8.1 HD 50
	Embryo-larvae	4-day LC50, 8.5 μg/l	F	20–24	pH 7.6–8.1 HD 200
	Largemouth bass (*Micropterus salmoides*)				
	Embryo-larvae	4-day LC50, 1.5 μg/l	F	20–24	pH 7.6–8.1 HD 50
	Embryo-larvae	4-day LC50, 1.8 μg/l	F	20–24	pH 7.6–8.1 HD 200
Capryl alcohol[200]	Rainbow trout				
	Fry	96-hour LC50, 75 mg/l	S	10.5	pH 8.2 HD 340
Carbaryl (99.2%)[153]	Medaka (*Oryzias latipes*)				
	Embryos	EC50, 2.5 mg/l	S	—	—
Carbaryl (94%)[127]	Carp (*Cyprinus carpio*)	96-hour LC50, 1.7 mg/l	S	29	pH 7.0 HD 22
Caustic (Celanese NaOH)[200]	Rainbow trout				
	Fry	96-hour LC50, 110 mg/l	S	10.5	pH 8.2 HD 340

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Chlordane[200]	Fathead minnow	32-day BCF, 37 800	F	25	pH 7.5 HD 45.5
Chlorinated paraffin[200]	Fathead minnow	32-day BCF, 49	F	25	pH 7.5 HD 45.5
Chlorine[229]	Golden shiner	Avoidance, .015–.017 mg/l	—	12–30	—
o-Chlorobenzaldehyde[285]	Rainbow trout Juvenile	96-hour LC50, 2.45 mg/l	S	15.2	pH 7.1–7.4 HD 290
4-Chlorobiphenyl[206]	Golden orfe (*Leuciscus idus melanotus*)	20-day BCF, 5 250	F	19	—
	Carp	20-day BCF, 890	F	25	—
	Brown trout (*Salmo trutta*)	20-day BCF, 4 025	F	19	—
	Guppy (*Poecilia reticulata*)	20-day BCF, 1 816	F	25	—
1-Chloro-3-bromopropane[282]	Goldfish	24-hour LC50, 75 mg/l	S	20	pH 6–8
2-Chlorophenol (2-CP)[288]	Goldfish	24-hour LC50, 16 mg/l	R	20	—
4-Chlorophenol (4-CP)[280]	Goldfish	24-hour LC50, 9.0 mg/l	R	20	—
3-Chloro-1,2-propanediol[282]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
3-Chloro-1-propanol[282]	Goldfish	24-hour LC50, 170 mg/l 96-hour LC50, 50 mg/l	S S	20 20	pH 6–8
Chromalit (potassium chromic sulfate)[290]	Rainbow trout Fry	96-hour LC50, 750 mg/l	S	10.5	pH 8.2 HD 340
CMC (sodium carboxymethyl cellulose)[280]	Rainbow trout Fry	96-hour LC50, >10 000 mg/l	S	10.5	pH 8.2 HD 340
Copper[240]	Rainbow trout	10-day reduced growth, 168 µg/l	S	12	pH 6.0 HD 365
		10-day reduced growth, 23 µg/l	S	12	pH 7.5 HD 30
		30-day reduced growth, 30 µg/l	S	12	
Copper[200]	Steelhead trout Adult male	96-hour LC50, 57 µg/l	F	9.2	pH 7.57 HD 42
	Coho salmon Adult male	96-hour LC50, 46 µg/l	F	9.4	pH 7.29 HD 20
Copper[205]	Cutthroat trout (*Salmo clarki*)	96-hour LC50, .02–.37 mg/l	F	12–16	—
	Rainbow trout	96-hour LC50, .08–.51 mg/l	F	12–16	—
Copper[206]	Sockeye salmon Fry	96-hour LC50, 210–220 µg/l	R	5–10	pH 6.1–6.9 HD 36–46
	Fingerling	96-hour LC50, 240 µg/l	R	14–17	pH 6.0–8.0
	Smolt	96-hour LC50, 240 µg/l	R	6.5–12	HD 36–46
Copper[204]	Sockeye salmon				
	Egg	Prehatch LC50, 37–78 µg/l	F	5–8	pH 7.6
	Fry at feeding	96-hour LC50, 110 µg/l	F	5–8	HD 83
	Fry	96-hour LC50, 150–170 µg/l	F	5–8	
	Smolt	96-hour LC50, 190–240 µg/l	F	5–8	
	Pink salmon				
	Fry at feeding	96-hour LC50, 8 µg/l	F	5–8	
	Fry	96-hour LC50, 200 µg/l	F	5–8	
Copper[206]	Bluegill (*Lepomis macrochirus*)				
	Juvenile	14-day LC50, 2.5 mg/l	—	24	pH 8.2
	Channel catfish	14-day LC50, 1.2 mg/l	—	22.5	HD 260

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Chromium (Cr^{6+})[40]	Fathead minnow				
	Juvenile	96-hour LC50, 33.2 mg/l	F	25	pH 7.8 HD 220
Crude oil[201]	Dolly varden				
	Juvenile	96-hour LC50, 2.8 mg/l	S	8	—
	Smolt	96-hour LC50, 2.7 mg/l	S	8	—
	Chinook salmon				
	Juvenile	96-hour LC50, 3.6 mg/l	S	6–9	—
	Coho salmon				
	Juvenile	96-hour LC50, 3.7 mg/l	S	8–9	—
	Embryo	96-hour LC50, >12.0 mg/l	S	8–9	—
	Alevin	96-hour LC50, >12.0 mg/l	S	8–9	—
	Emergent fry	96-hour LC50, 8.0 mg/l	S	8–9	—
	Sockeye salmon				
	Juvenile	96-hour LC50, 1.79 mg/l	S	6	—
	Smolt	96-hour LC50, 2.2 mg/l	S	6	—
	Arctic grayling				
	Juvenile	96-hour LC50, 4.4 mg/l	S	9	—
	Arctic char				
	Juvenile	96-hour LC50, 2.2 mg/l	S	4	—
	Slimy sculpin	96-hour LC50, 6.4 mg/l	S	9	—
	Threespine stickleback	96-hour LC50, >10.5 mg/l	S	5–8	—
	Pink salmon				
	Embryo	96-hour LC50, >12.0 mg/l	S	4	—
	Alevin	96-hour LC50, >12.0 mg/l	S	4	—
	Emergent fry	96-hour LC50, 8.0 mg/l	S	4	—
	Outmigrant fry	96-hour LC50, 8.0 mg/l	S	4	—
CS gas (ortho-chlorobenzylidene malononitrile)[205]	Rainbow trout				
	Juvenile	96-hour LC50, 0.22 mg/l	S	15.2	pH 7.1–7.4 HD 290
Cupric acetate[204]	Fathead minnow				
	Juvenile	96-hour LC50, 0.39 mg/l	S	22	pH 7.2–7.9 HD 40–48
Cyanide (HCN)[39]	Largemouth bass	96-hour LC50, 101 μg/l	F	25	pH 7.9
	Black crappie (*Pomoxis nigromaculatus*)	96-hour LC50, 101 μg/l	F	25	pH 7.9
1,5,9-Cyclododecatriene[202]	Goldfish	24-hour LC50, 4 mg/l	S	20	pH 6–8
1,3,5-Cycloheptatriene[202]	Goldfish	24-hour LC50, 15 mg/l	S	20	pH 6–8
1,5-Cyclooctadiene[202]	Goldfish	24-hour LC50, 14 mg/l	S	20	pH 6–8
Cypermethrin (tech. grade)[104]	Rainbow trout	24-hour LC50, 55 μg/l	S	10	pH 7.5 HD 110
Cypermethrin (40% EC)[104]	Rainbow trout	24-hour LC50, 11 μg/l	S	10	pH 7.5 HD 110
2,4-D (74.7%)[188]	Bleak (*Alburnus alburnus*)				
	Embryo	48-hour LC50, 12.9 mg/l	S	23	pH 8.4
	Larvae	48-hour LC50, 51.6 mg/l	S	23	
2,4-D (amine)[189]	Mosquitofish	96-hour LC50, 405 mg/l	S	21–22	—
2,4-D (BE)[186]	Cutthroat trout	96-hour LC50, 490 μg/l	S	5	pH 7.2
		96-hour LC50, 770 μg/l	S	15	HD 40
	Lake trout (*Salvelinus namaycush*)	96-hour LC50, 600 μg/l	S	5	
		96-hour LC50, 820 μg/l	S	15	
	Cutthroat trout	96-hour LC50, 540–860 μg/l	S	10	pH 6.5–8.5
	Lake trout	96-hour LC50, 640–1 170 μg/l	S	10	HD 40–300
	Cutthroat trout				
	Embryo-larvae	61-day MATC,[a] 44–24 μg/l	F	11	pH 7.4 HD 162
	Lake trout				
	Embryo-larvae	61-day MATC, 33–15 μg/l	F	11	

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
2,4-D (PGBEE)[104]	Cutthroat trout	96-hour LC50, 490 µg/l	S	5	pH 7.2
		96-hour LC50, 780 µg/l	S	15	HD 40
	Lake trout	96-hour LC50, 700 µg/l	S	5	
		96-hour LC50, 1 000 µg/l	S	15	
	Cutthroat trout	96-hour LC50, 930–1 220 µg/l	S	10	pH 6.5–8.5
	Lake trout	96-hour LC50, 630–1 200 µg/l	S	10	HD 40–300
	Cutthroat trout Embryo-larvae	61-day MATC, 60–31 µg/l	F	11	pH 7.4 HD 162
	Lake trout Embryo-larvae	61-day MATC, 100–52 µg/l	F	11	
p,p'-DDE[300]	Fathead minnow	32-day BCF, 51 000	F	25	pH 7.5 HD 45.5
o,p'-DDT[300]	Fathead minnow	32-day BCF, 37 000	F	25	pH 7.5 HD 45.5
p,p'-DDT[300]	Fathead minnow	32-day BCF, 29 400	F	25	pH 7.5 HD 45.5
Decamethrin (NRDC 161)[105]	Atlantic salmon (*Salmo salar*)	96-hour lethal threshold, 0.52 µg/l	S	10	—
Decamethrin (NRDC 168S)[105]	Atlantic salmon	96-hour lethal threshold, 1.45 µg/l	S	10	—
Decis (25% EC)[105]	Atlantic salmon	96-hour lethal threshold, 0.75 µg/l	S	10	—
Demicron[101]	Loach (*Lepidocephalus thermalis*)	48-hour LC50, 44 mg/l	—	—	—
Diacetone alcohol[302]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Diallylamine[302]	Goldfish	96-hour LC50, 20 mg/l	S	20	pH 7.0
		96-hour LC50, 7 mg/l	S	20	pH 9.4
Diazinon[110]	Silver crucian carp (*Cyrpinus auratus*)	7-day BCF, 36.6	F	20	pH 6.8
	Carp	7-day BCF, 65.1	F	20	
	Guppy (*Labistes reticulata*)	7-day BCF, 17.5	F	20	
	Topmouth gudgeon (*Pseudorasbora parva*)	7-day BCF, 152	F	20	
		14-day BCF, 150	F	20	
		7-day BCF, 206	F	20	
Diazinon (2% dust)[109]	Bluegill	96-hour LC50, 0.17 mg/l	S	20	pH 8.2 HD 192
Diazinon (48.2% EC)[109]	Fathead minnow	96-hour LC50, 3.7 mg/l	S	20	pH 8.2
	Bluegill	96-hour LC50, 0.53 mg/l	S	20	HD 192
Dicamba (Banex)[139]	Mosquitofish	96-hour LC50, 465 mg/l	S	21–22	—
o-Dichlorobenzene[304]	Fathead minnow Juvenile	96-hour LC50, 57.0 mg/l	S	22	pH 7.2–7.9 HD 40–48
p-Dichlorobenzene[304]	Fathead minnow Juvenile	96-hour LC50, 33.7 mg/l	S	22	pH 7.2–7.9 HD 40–48
2,4-Dichlorophenol (2,4-CP)[288]	Goldfish	24-hour LC50, 7.8 mg/l	R	20	—
1,3-Dichloropropane[302]	Goldfish	24-hour LC50, 160 mg/l	S	20	pH 6–8
1,3-Dichloro-2-propanol[302]	Goldfish	24-hour LC50, 680 mg/l	S	20	pH 6–8
Diethanolamine[302]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 7.0
		24-hour LC50, 800 mg/l	S	20	pH 9.7
Diethylene glycol[302]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Diethylene glycol mono-n-butyl ether[302]	Goldfish	24-hour LC50, 2 700 mg/l	S	20	pH 6–8
Diethylene glycol monomethyl ether[302]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Diethyl ketone[302]	Goldfish	24-hour LC50, 1 200 mg/l	S	20	pH 6–8

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
2-(Digeranylamino)-ethanol[160]	Fathead minnow	96-hour LC50, .073–.547 µl/l	S	7–22	pH 6.5–9.5
Diisononylphthalate (DINP)[207]	Channel catfish				
	Embryo-larvae	4-day LC50, 0.42 mg/l	R	29–31	pH 7.6–8.1 HD 90–115
	Redear sunfish				
	Embryo-larvae	4-day LC50, 4.67 mg/l	R	20–24	
Diisopropanolamine[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 7.0
		24-hour LC50, 1 100 mg/l	S	20	pH 9.7
Diisopropyl ether[202]	Goldfish	24-hour LC50, 380 mg/l	S	20	pH 6–8
Diisopropylnaphthalene (DIPN)[212]	Carp	28-day BCF, 203	R	25	—
Dimethylamine[204]	Rainbow trout	96-hour LC50, 120 mg/l	S	15	pH 7.2
		50-day LC50, 1.2 mg/l	F	15	HD 320
	Embryo-larvae	50-day MATC, 0.6 mg/l	F	15	
		96-hour LC50, 20 mg/l	S	15	pH 7.2 HD 20
Dioctylphthalate (DOP)[207]	Channel catfish				
	Embryo-larvae	4-day LC50, 0.69 mg/l	R	29–31	pH 7.6–8.1 HD 90–115
	Redear sunfish				
	Embryo-larvae	4-day LC50, 6.18 mg/l	R	20–24	
	Rainbow trout				
	Embryo-larvae	4-day LC50, 139.5 mg/l	F	13.5–14.3	pH 7.6–8.1 HD 50
	Embryo-larvae	4-day LC50, 149.2 mg/l	F	13.5–14.3	pH 7.6–8.1 HD 200
	Largemouth bass				
	Embryo-larvae	4-day LC50, 42.1 mg/l	F	20–24	pH 7.6–8.1 HD 50
	Largemouth bass				
	Embryo-larvae	4-day LC50, 32.9 mg/l	F	20–24	pH 7.6–8.1 HD 200
Diphenylamine[200]	Fathead minnow	32-day BCF, 30	F	25	pH 7.5 HD 45.5
Dipropylene glycol[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Dissolved oxygen (DO)[26]	Atlantic salmon				
	Smolt	3-day LC50, 3 mg/l	S	13–15	pH 7.6–8.1 HD 250
Diquat[127]	Carp	96-hour LC50, 50.3 mg/l	S	29	pH 7.0 HD 22
β-n-Dodecenylsuccinic anhydride[202]	Goldfish	96-hour LC50, 3 mg/l	S	20	pH 7.0
		96-hour LC50, 2 mg/l	S	20	pH 6.0
Dominion Rigwash (surfactant)[220]	Rainbow trout				
	Fry	96-hour LC50, 13 mg/l	S	10.5	pH 8.2 HD 340
2,2-DPA (Dowpon)[150]	Mosquitofish	96-hour LC50, 19 100 mg/l	S	21–22	—
Drilling fluid (used)[220]	Rainbow trout				
	Fry	96-hour LC50, 0.83–5.3 percent	S	10.5	pH 8.2 HD 340
Electroconductive resin (ECR 77)[234]	Carp	48-hour LC50, 27.2 mg/l	—	—	—
	Rainbow trout	48-hour LC50, 4.2 mg/l	—	—	—
	Guppy	48-hour LC50, 3.7 mg/l	—	—	—
Endrin[101]	Loach	48-hour LC50, .003 mg/l	—	—	—
Epichlorohydrin[202]	Goldfish	24-hour LC50, 23 mg/l	S	20	pH 6–8
Ethyl amyl ketone[202]	Goldfish	24-hour LC50, 80 mg/l	S	20	pH 6–8
Ethylene glycol[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Ethylene glycol mono-n-butyl ether[202]	Goldfish	24-hour LC50, 1 700 mg/l	S	20	pH 6–8
Ethylene glycol monoethyl ether (2 ethoxyethanol)[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Ethylene glycol monoethyl ether (2 methoxyethanol)[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Ethylene glycol monoethyl ether acetate[202]	Goldfish	24-hour LC50, 160 mg/l	S	20	pH 6–8
Ethylene glycol monomethyl ether acetate[202]	Goldfish	24-hour LC50, 190 mg/l	S	20	pH 6–8
Ethylene glycol mono-i-propyl ether[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Ethylene oxide[202]	Goldfish	24-hour LC50, 90 mg/l	S	20	pH 6–8
2-Ethylhexanol[207]	Rainbow trout Fry	120-hour LC50, <75 mg/l	S	20	pH 7.0 HD 180
2-Ethylhexyl glycidyl ether[202]	Goldfish	24-hour LC50, 14 mg/l	S	20	pH 6–8
Ethyl parathion (50% EC)[102]	Khard	96-hour LC50, 1.8 to 4.9 mg/l	S	17–32	pH 6.6–7.8 HD 60–108
	Patra	96-hour LC50, 0.42 to 1.35 mg/l	S	17–32	pH 6.6–7.8 HD 60–180
Fenoprop (Farmco TP-20)[102]	Mosquitofish	96-hour LC50, 0.35 mg/l	S	21–22	—
Fenpropanate (tech. grade)[110]	Rainbow trout	24-hour LC50, 76.7 µg/l	S	10	pH 7.5 HD 110
Fenpropanate (2.5% EC)[104]	Rainbow trout	24-hour LC50, 8.6 µg/l	S	10	pH 7.5 HD 110
Fenvalerate (tech. grade)[104]	Rainbow trout	24-hour LC50, 76 µg/l	S	10	pH 7.5 HD 110
Fenvalerate (30% EC)[104]	Rainbow trout	24-hour LC50, 21 µg/l	S	10	pH 7.5 HD 110
Ferrochrome lignosulphonate[200]	Rainbow trout Fry	96-hour LC50, 1 500 mg/l	S	10.5	pH 8.2 HD 340
Fire-Trol 931[207]	Rainbow trout Juvenile	96-hour LC50, 1 238 mg/l	F	10–20	pH 7
	Juvenile	96-hour LC50, 150 mg/l	F	10–20	pH 8
FLR-100 (fluid loss reducer)[200]	Rainbow trout Fry	96-hour LC50, >1 000 mg/l	S	10.5	pH 8.2 HD 340
Fluoranthene[229]	Rainbow trout	21-day BCF, 378	F	—	pH 7.0 HD 45
Glycerin[207]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Glycerin-α-allyl ether[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Glycidyltrimethylammonium chloride[202]	Goldfish	24-hour LC50, 5 000 mg/l	S	20	pH 6–8
Glycol[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Glyphosphate[201]	Rainbow trout	96-hour LC50, 140–240 mg/l	S	12	pH 6.5–9.5
	Bluegill	96-hour LC50, 140–220	S	22	HD 40
	Fathead minnow	96-hour LC50, 97 mg/l	S	22	pH 7.2
	Channel catfish	96-hour LC50, 130 mg/l	S	22	HD 40
α-HCH[208]	Golden orfe	20-day BCF, 1 216	F	19	—
	Carp	20-day BCF, 330	F	25	—
	Brown trout	20-day BCF, 605	F	19	—
	Guppy	20-day BCF, 586	F	25	—
β-HCH[208]	Golden orfe	20-day BCF, 973	F	19	—
	Carp	20-day BCF, 273	F	25	—
	Brown trout	20-day BCF, 658	F	19	—
	Guppy	20-day BCF, 1 485	F	25	—
γ-HCH[208]	Golden orfe	20-day BCF, 765	F	19	—

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
	Carp	20-day BCF, 281	F	25	—
	Brown trout	20-day BCF, 442	F	19	—
	Guppy	20-day BCF, 938	F	25	—
HCN[40]	Fathead minnow				
	Juvenile	96-hour LC50, 0.125 mg/l	F	25	pH 7.8 HD 220
	Rainbow trout				
	Juvenile	96-hour LC50, 0.057 mg/l	F	10	pH 7.95 HD 220
HDEHP[297]	Rainbow trout				
	Fry	120-hour LC50, 200–340 mg/l	S	10–20	pH 7.0 HD 180
Heptachlor[300]	Fathead minnow	32-day BCF, 9 500	F	25	pH 7.5 HD 45.5
Heptachlorepoxide[300]	Fathead minnow	32-day BCF, 14 400	F	25	pH 7.5 HD 45.5
Heptachloronorbornene[300]	Fathead minnow	32-day BCF, 11 100	F	25	pH 7.5 HD 45.5
Heptachloronorbornene[292]	Fathead minnow				
	Larvae-juvenile	96-hour LC50, 85.6 µg/l	F	25	pH 7.2–7.7
	Larvae-juvenile	30-day LC50, 60.1 µg/l	F	25	HD 42–43
	Larvae-juvenile	30-day BCF, 11 200	F	25	
Hexabromobiphenyl[300]	Fathead minnow	32-day BCF, 18 100	F	25	pH 7.5 HD 45.5
Hexabromocyclododecane[300]	Fathead minnow	32-day BCF, 18 100	F	25	pH 7.5 HD 45.5
Hexachlorobenzene[300]	Fathead minnow	32-day BCF, 18 500	F	25	pH 7.5 HD 45.5
Hexachlorocyclopentadiene[300]	Fathead minnow	32-day BCF, 29	F	25	pH 7.5 HD 45.5
Hexachlorocyclopentadiene[292]	Fathead minnow				
	Larvae-juvenile	96-hour LC50, 7.0 µg/l	F	25	pH 7.2–7.7
	Larvae-juvenile	30-day LC50, 6.7 µg/l	F	25	HD 42–43
	Larvae-juvenile	30-day BCF, <11	F	25	
Hexachloronorbornadiene[300]	Fathead minnow	32-day BCF, 6 400	F	25	pH 7.5 HD 45.5
Hexachloronorbornadiene[292]	Fathead minnow				
	Larvae-juvenile	96-hour LC50, 188 µg/l	F	25	pH 7.2–7.7
	Larvae-juvenile	30-day LC50, 123 µg/l	F	25	HD 42–43
	Larvae-juvenile	30-day BCF, 6 400	F	25	
Hexylene glycol[302]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Kelzen-XC (Xanthate gum)[220]	Rainbow trout				
	Fry	96-hour LC50, 420 mg/l	S	10.5	pH 8.2 HD 340
Kerosene[293]	Fathead minnow	96-hour mortality, 8 980 mg/l	F	25	—
		24-hour mortality, 5 000 mg/l	S	25	—
Lead[215]	*Colisa fasciatus*	96-hour LC50, 19 mg/l	S	29	pH 7.3 HD 120
Lead (lead nitrate)[257]	*Channa punctatus*	96-hour LC50, 13.2 mg/l	S	—	—
Lindane[300]	Fathead minnow	32-day BCF, 180	F	25	pH 7.5 HD 45.5
Lindane (99%)[127]	Carp	96-hour LC50, 0.20 mg/l	S	29	pH 7.0 HD 22

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
LIX 64 N[297]	Rainbow trout				
	Fry	120-hour LC50, 240–490 mg/l	S	10–20	pH 7.0 HD 180
Malathion[121]	Loach	48-hour LC50, 21 mg/l	—	—	—
Malononitrile[295]	Rainbow trout				
	Juvenile	96-hour LC50, 1.63 mg/l	S	15.2	pH 7.1–7.4 HD 290
Mercury[204]	Sockeye salmon				
	Eggs	Prehatch LC50, 4–5 µg/l	F	5–8	pH 7.6
	Fry (first feeding)	168-hour LC50, 280–300 µg/l	F	5–8	HD 83
	Fry (older)	168-hour LC50, 180–220 µg/l	F	5–8	
	Smolt	168-hour LC50, 180–220 µg/l	F	5–8	
	Pink salmon				
	Egg	Prehatch LC50, 4–5 µg/l	F	5–8	
	Fry (first feeding)	168-hour LC50, 140 µg/l	F	5–8	
	Fry (older)	168-hour LC50, 210 µg/l	F	5–8	
Mercury[212]	Goldfish				
	Juvenile	48-hour LC50, 0.33–0.38 mg/l	S	25	HD 100
Mercury (mercuric acetate)[204]	Fathead minnow	96-hour LC50, 0.19 mg/l	S	22	pH 7.2–7.9 HD 40–48
Mercury (mercuric chloride)[213]	*Channa punctatus*	96-hour LC50, 1.8 mg/l	S	—	—
Mercury (mercuric thiocyanate)[204]	Fathead minnow				
	Juvenile	96-hour LC50, 0.15 mg/l	S	22	pH 7.2–7.9 HD 40–48
Mesityl oxide[202]	Goldfish	24-hour LC50, 540 mg/l	S	20	pH 6–8
Metacid[121]	Loach	48-hour LC50, 5.8 mg/l	—	—	—
Methallyl chloride[202]	Goldfish	24-hour LC50, 14 mg/l	S	20	pH 6–8
Methamidophos (90%)[127]	Carp	96-hour LC50, 69 mg/l	S	29	pH 7.0 HD 22
Methoxychlor[200]	Fathead minnow	32-day BCF, 8 300	F	25	pH 7.5 HD 45.5
4-Methoxy-4-methyl-2-pentanone[202]	Goldfish	24-hour LC50, 3 800 mg/l	S	20	pH 6–8
Methyl ethyl ketone[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Methylisobutylcarbinol[202]	Goldfish	24-hour LC50, 360 mg/l	S	20	pH 6–8
Methyl isobutyl ketone[202]	Goldfish	24-hour LC50, 460 mg/l	S	20	pH 6–8
2-Methyl-2,4-pentanediol[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Metso-Beads[200]	Rainbow trout				
	Fry	96-hour LC50, 240 mg/l	S	10.5	pH 8.2 HD 340
Mirex[200]	Fathead minnow	32-day BCF, 18 100	F	25	pH 7.5 HD 45.5
Monoallylamine[202]	Goldfish	96-hour LC50, 27 mg/l	S	20	pH 7.0
		96-hour LC50, 6 mg/l	S	20	pH 9.5
Monoethanolamine[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 7.0
		96-hour LC50, 170 mg/l	S	20	pH 10.0
Monoisopropanolamine[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 7.0
		96-hour LC50, 210 mg/l	S	20	pH 9.9
NA-SUL AS-50[297]	Rainbow trout				
	Fry	120-hour LC50, <32 mg/l	S	20	pH 7.0 HD 180
Nickel[214]	Carp				
	Larvae	72-hour LC50, 8.46 mg/l	R	23–25	pH 7.4
	Larvae	96-hour LC50, 6.16 mg/l	R	23–25	HD 128

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
β-n-Nonenylsuccinic anhydride[202]	Goldfish	96-hour LC50, 250 mg/l	S	20	pH 7.0
		96-hour LC50, 10 mg/l	S	20	pH 5.0
i-Nonyl alcohol[203]	Goldfish	24-hour LC50, 16 mg/l	S	20	pH 6–8
Octachlorostyrene[204]	Fathead minnow	32-day BCF, 33 000	F	25	pH 7.5 HD 45.5
Ozone[20]	Rainbow trout Juvenile	96-hour LC50, 9.3 μg/l	F	10	pH 6.8 HD 20
Ozone[21]	Bluegill	24-hour LC50, 0.06 mg/l	F	10	pH 7.1
Ozone produced oxidant[25]	White perch (*Morone americana*)	96-hour LC50, 0.22 mg/l	F	15	—
Paraformaldehyde[205]	Rainbow trout Fry	96-hour LC50, 60 mg/l	S	10.5	pH 8.2 HD 340
Paraquat[150]	Mosquitofish	96-hour LC50, 604 mg/l	S	21–22	—
Parathion[101]	Leech	48-hour LC50, 1.3 mg/l	—	—	—
PBB[174]	Fathead minnow	14-day BCF, >10 000	—	—	—
PCB:					
Aroclor 1016[206]	Fathead minnow	32-day BCF, 42 500	F	25	pH 7.5 HD 45.5
Aroclor 1060[206]	Fathead minnow	32-day BCF, 194 000	F	25	pH 7.5 HD 45.5
Aroclor 1248[206]	Fathead minnow	32-day BCF, 70 500	F	25	pH 7.5 HD 45.5
Aroclor 1254[206]	Fathead minnow	32-day BCF, 100 000	F	25	pH 7.5 HD 45.5
PCB:					
Aroclor 1016[207]	Channel catfish Embryo-larvae	4-day LC50, 11.16 μg/l	R	29–31	pH 7.6–8.1 HD 90–115
	Goldfish Embryo-larvae	4-day LC50, 13.21 μg/l	R	20–24	
	Rainbow trout Embryo-larvae	4-day LC50, 1.08 μg/l	R	13.5–14.3	
	Redear sunfish Embryo-larvae	4-day LC50, 7.82 μg/l	R	20–24	
Aroclor 1242[207]	Channel catfish Embryo-larvae	4-day LC50, 4.24 μg/l	R	29–31	pH 7.6–8.1 HD 90–115
	Goldfish Embryo-larvae	4-day LC50, 2.64 μg/l	R	20–24	
	Rainbow trout Embryo-larvae	4-day LC50, 1.03 μg/l	R	13.5–14.3	
	Redear sunfish Embryo-larvae	4-day LC50, 3.56 μg/l	R	20–24	
Aroclor 1254[207]	Channel catfish Embryo-larvae	4-day LC50, 1.76 μg/l	R	29–31	pH 7.6–8.1 HD 90–115
	Goldfish Embryo-larvae	4-day LC50, 1.18 μg/l	R	20–24	
	Rainbow trout Embryo-larvae	4-day LC50, 0.32 μg/l	R	13.5–14.3	
	Redear sunfish Embryo-larvae	4-day LC50, 0.53 μg/l	R	20–24	
Pentachlorophenol[208]	Fathead minnow	32-day BCF, 770	F	25	pH 7.5 HD 45.5
Pentachlorophenol[175]	Bluegill	96-hour LC50, 0.3 mg/l	S	—	pH 7.4
Pentachlorophenol[209]	Goldfish	24-hour LC50, 0.27 mg/l	R	20	—
Pentaerythritol triallyl ether[210]	Goldfish	96-hour LC50, 70 mg/l	S	20	pH 6–8
Permethrin[158]	Channel catfish	96-hour LC50, 1.10 μg/l	S	24	pH 8.4
	Mosquitofish	96-hour LC50, 15 μg/l	S	24	HD 100
	Largemouth bass	96-hour LC50, 8.5 μg/l	S	24	

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Permethrin (tech. grade)[104]	Rainbow trout	24-hour LC50, 135 µg/l	S	10	pH 7.5 HD 110
Permethrin (50% EC)[104]	Rainbow trout	24-hour LC50, 61 µg/l	S	10	pH 7.5 HD 110
Permethrin[105]	Atlantic salmon	96-hour lethal threshold, 8.8 µg/l	S	10	—
Permethrin (NRDC 167)[105]	Atlantic salmon	96-hour lethal threshold, 0.94 µg/l	S	10	—
Phenol[289]	Rainbow trout	48-hour LC50, 9.0–10.4 mg/l 48-hour LC50, 7.7 mg/l (pre-exposure to 0.04 mg Cl_2/l)	F F	15 15	pH 7.4–7.7 HD 75–89
Phenol[289]	Goldfish	24-hour LC50, 60 mg/l	R	20	—
Phenol[292]	Goldfish	24-hour LC50, 46 mg/l	S	20	pH 6–8
Phenyl glycidyl ether[292]	Goldfish	96-hour LC50, 43 mg/l	S	20	pH 6–8
n-Phenyl-2-naphylamine[289]	Fathead minnow	32-day BCF, 147	F	25	pH 7.5 HD 45.5
Phorone[292]	Goldfish	24-hour LC50, 60 mg/l	S	20	pH 6–8
Phos-Chek XA (fire retardant)[297]	Rainbow trout				
	Juvenile	96-hour LC50, 224 mg/l	F	10–20	pH 7
	Juvenile	96-hour LC50, 139 mg/l	F	10–20	pH 8
Phosphorus (P_4)[289]	Mosquitofish	96-hour LC50, 75 µg/l	S	20.5	pH 7.3 HD 55
	Bluegill	96-hour LC50, 29 µg/l	S	20.5	pH 7.6 HD 50–60
Picloram[110]	Mosquitofish	96-hour LC50, 120 mg/l	S	21–22	—
Pivalic acid[292]	Goldfish	24-hour LC50, 4 500 mg/l 96-hour LC50, 380 mg/l	S S	20 20	pH 7.0 pH 5.4
Platinum[216]	Coho salmon				
	Juvenile	24-hour LC50, 15.5 mg/l	R	8.5	pH 6.5
	Juvenile	48-hour LC50, 5.2 mg/l	R	8.5	HD 55
	Juvenile	96-hour LC50, 2.5 mg/l	R	8.5	
Polyethylene glycol[292]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Pounce (synthetic pyrethroid)[107]	Channel catfish	96-hour LC50, 1.1 µg/l	S	24	pH 8.4
	Mosquitofish	96-hour LC50, 13 µg/l	S	24	HD 100
	Largemouth bass	96-hour LC50, 8.5 µg/l	S	24	
Primene JM-T[297]	Rainbow trout				
	Fry	120-hour LC50, 1.2–5.3 mg/l	S	10–20	pH 7.0 HD 180
1,2-Propanediol[292]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
1,3-Propanediol[292]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
i-Propyl alcohol[292]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Propylene glycol[292]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Propylene oxide[292]	Goldfish	24-hour LC50, 170 mg/l	S	20	pH 6–8
Pyrene[329]	Rainbow trout	21-day BCF, 72	F	10	pH 7.0 HD 45
Resorcinol[254]	Fathead minnow				
	Juvenile	96-hour LC50, 53.4 mg/l	S	22	pH 7.2–7.9 HD 40–48
Roundup[101]	Rainbow trout				
	Eyed eggs	96-hour LC50, 16 mg/l	S	12	pH 7.2
	Sac fry	96-hour LC50, 3.4 mg/l	S	12	HD 40
	Swim-up fry	96-hour LC50, 2.4 mg/l	S	12	
	Fingerlings (1.0g)	96-hour LC50, 1.3–14 mg/l	S	7–17	
	Fathead minnow	96-hour LC50, 1.0–2.3 mg/l	S	22	
	Channel catfish				
	Eyed eggs	24-hour LC50, 43 mg/l	S	22	

TABLE 1—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
	Sac fry	96-hour LC50, 4.3 mg/l	S	22	
	Swim-up fry	96-hour LC50, 3.3 mg/l	S	22	
	Fingerling (2.2g)	96-hour LC50, 13 mg/l	S	22	
	Bluegill	96-hour LC50, 3.0–7.5 mg/l	S	17–27	
SAPP (sodium acid pyrophosphate)[289]	Rainbow trout Fry	96-hour LC50, 840 mg/l	S	10.5	pH 8.2 HD 340
Skot-Free (surfactant)[290]	Rainbow trout Fry	96-hour LC50, 48 mg/l	S	10.5	pH 8.2 HD 340
Sodium hypochlorite[214]	Fathead minnow Juvenile	96-hour LC50, 5.9 mg/l	S	22	pH 7.2–7.9 HD 40–48
Sodium pentachlorophenate[291]	Chinook salmon Juvenile	96-hour LC50, 0.078 mg/l	F	12	pH 7.0–7.1 HD 54
SS-100[290]	Rainbow trout Fry	96-hour LC50, >1 000 mg/l	S	10.5	pH 8.2 HD 340
STAFLO (fluid loss reducer)[290]	Rainbow trout Fry	96-hour LC50, >1 000 mg/l	S	10.5	pH 8.2 HD 340
Styrene[292]	Goldfish	24-hour LC50, 26 mg/l	S	20	pH 6–8
Sulfolane[282]	Goldfish	24-hour LC50, 4 800 mg/l 24-hour LC50, 140 mg/l	S S	20 20	pH 6–8
Swascofix[220]	*Mystus vittatus*	15-day ATPase reduction, 0.6 mg/l 30-day ATPase reduction, 0.6 mg/l	S S	— —	— —
Sw.ft's Rigwash (surfactant)[290]	Rainbow trout Fry	96-hour LC50, 19 mg/l	S	10.5	pH 8.2 HD 340
Tafazine[160]	*Sarotherodon mossambicus* *Puntius ticto* *Danio* sp.	96-hour LC50, 3.1 mg/l 96-hour LC50, 24.5 mg/l 96-hour LC50, 12.6 mg/l	S S S	28.5 28.5 28.5	pH 8.5
2,4,5-T amine[159]	Mosquitofish	96-hour LC50, 1 600 mg/l	S	21–22	—
TBP[287]	Rainbow trout Fry	120-hour LC50, 42–105 mg/l	S	10–20	pH 7.0 HD 180
TCDD[308]	Mosquitofish Channel catfish Fingerling	7 to 14-day BCF, 6 400 6-day BCF, 2 200	S S	— —	— —
Tert-butyl alcohol[282]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
p-Tert-butylbenzoic acid[289]	Goldfish	96-hour LC50, 33 mg/l 96-hour LC50, 4 mg/l	S S	20 20	pH 7.0 pH 5.0
p-Tert-butyltoluene[282]	Goldfish	24-hour LC50, 3 mg/l	S	20	pH 6–8
2,3,4,5-Tetrachlorobiphenyl[306]	Golden orfe Carp Brown trout Guppy	20-day BCF, 19 300 20-day BCF, 3 750 20-day BCF, 8 750 20-day BCF, 8 000	F F F F	19 25 19 25	— — — —
3,4,3',4'-Tetrachlorobiphenyl[306]	Golden orfe Carp Brown trout Guppy	20-day BCF, 7 860 20-day BCF, 1 750 20-day BCF, 4 300 20-day BCF, 14 200	F F F F	19 25 19 25	— — — —
2,3,4,6-Tetrachlorophenol[288]	Goldfish	24-hour LC50, 0.75 mg/l	R	20	—
Toluene[282]	Goldfish	24-hour LC50, 58 mg/l	S	20	pH 6–8
Toluene diamine[300]	Fathead minnow	32-day BCF, 91	F	25	pH 7.5 HD 45.5

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Toluene-2,4-diisocyanate[204]	Fathead minnow Juvenile	96-hour LC50, 164.5 mg/l	S	22	pH 7.2–7.9 HD 40–48
Torq-trim (triglycerides and alcohols)[280]	Rainbow trout Fry	96-hour LC50, 1 300 mg/l	S	10.5	pH 8.2 HD 340
Toxaphene[114]	Bluegill	96-hour LC50, 5.0–7.8 µg/l	S	18	pH 7.2–7.6 HD 40–48
		32-day BCF, 4 700–8 100	S	—	—
Toxaphene (Fraction 1)[114]	Bluegill	96-hour LC50, 25 µg/l	S	18	pH 7.2–7.6 HD 40–48
		32-day BCF, 600–840	S	—	—
Toxaphene (Fraction 2)[114]	Bluegill	96-hour LC50, 17 µg/l	S	18	pH 7.2–7.6 HD 40–48
Toxaphene (Fraction 3)[114]	Bluegill	96-hour LC50, 7.3 µg/l	S	18	pH 7.2–7.6 HD 40–48
Toxaphene (Fraction 4)[114]	Bluegill	96-hour LC50, 5.7 µg/l	S	18	pH 7.2–7.6 HD 40–48
Toxaphene (Fraction 5)[114]	Bluegill	96-hour LC50, 3.4 µg/l	S	18	pH 7.2–7.6 HD 40–48
Toxaphene (Fraction 6)[114]	Bluegill	96-hour LC50, 2.6 µg/l	S	18	pH 7.2–7.6 HD 40–48
Toxaphene (Fraction 7)[114]	Bluegill	96-hour LC50, 7.8 µg/l	S	18	pH 7.2–7.6 HD 40–48
Toxaphene (Fraction 8)[114]	Bluegill	96-hour LC50, 39 µg/l	S	18	pH 7.2–7.6 HD 40–48
Toxaphene (Fraction 9)[114]	Bluegill	96-hour LC50, 28 µg/l	S	18	pH 7.2–7.6 HD 40–48
	Bluegill	32-day BCF, 4 200–6 700	S	—	—
Transformer fluid (Dow Corning 561 silicone)[227]	Channel catfish Embryo-larvae	4-day LC50, 3.2 mg/l	R	29–31	pH 7.6–8.1 HD 90–115
	Redear sunfish Embryo-larvae	4-day LC50, 37.8 mg/l	R	20–24	
2,3,6-Tribromoanisole[201]	Fathead minnow	32-day BCF, 1 300	F	25	—
2,4,6-Tribromoanisole[200]	Fathead minnow	32-day BCF, 865	F	25	pH 7.5 HD 45.5
2,4,6-Tribromophenol[201]	Fathead minnow	32-day BCF, 83	F	25	—
Trichloroacetic acid[199]	Fathead minnow	96-hour LC50, 2 000 mg/l	S	20	pH 8.2 HD 192
Trichlorobenzene[210]	Rainbow trout	35-day BCF, 100–400	S and F	—	—
1,2,4-Trichlorobenzene[200]	Fathead minnow	32-day BCF, 2 800	F	25	pH 7.5 HD 45.5
2,4,5-Trichlorophenol[205]	Goldfish	24-hour LC50, 1.7 mg/l	R	20	—
2,4,6-Trichlorophenol[205]	Goldfish	24-hour LC50, 10 mg/l	R	20	—
Tricresyl phosphate[200]	Fathead minnow	32-day BCF, 165	F	25	pH 7.5 HD 45.5
Tri-Cron (propane & sulfonates)[280]	Rainbow trout Fry	96-hour LC50, 75 mg/l	S	10.5	pH 8.2 HD 340
Triethanolamine[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 9.9

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Triethylene glycol[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Triethylene glycol monoethyl ether[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Trifluralin[157]	Fathead minnow	40-hour BCF, 3 261	S	19.5–21.5	—
Trimethylene glycol[202]	Goldfish	24-hour LC50, >5 000 mg/l	S	20	pH 6–8
Tris (2,3-dibromopropyl) phosphate[200]	Fathead minnow	32-day BCF, 2.7	F	25	pH 7.5 HD 45.5
Trisodium carboxymethyloxy-succinate[222]	Goldfish	96-hour LC50, 2 900 mg/l	R	16–18	pH 7.8–8.2
	Rainbow trout	96-hour LC50, 2 300 mg/l	R	16–18	HD 300
Vanadium[217]	Flagfish (*Jordanella floridae*)				
	Adult	96-hour LC50, 11.2 mg/l	F	24	pH 8.2
	Larvae	28-day LC50, 1.13–1.88 mg/l	F	24	HD 275
Versatic 10[297]	Rainbow trout				
	Fry	120-hour LC50, 3–9 mg/l	S	10–20	pH 7.0 HD 180
m-Xylene[202]	Goldfish	24-hour LC50, 16 mg/l	S	20	pH 6–8
o-Xylene[202]	Goldfish	24-hour LC50, 13 mg/l	S	20	pH 6–8
p-Xylene[202]	Goldfish	24-hour LC50, 18 mg/l	S	20	pH 6–8
3,5-Xylenol[202]	Goldfish	96-hour LC50, 22 mg/l	S	20	pH 6–8
Zinc[40]	Fathead minnow				
	Juvenile	96-hour LC50, 2.61 mg/l	F	25	pH 7.8 HD 220
Zinc[200]	Fathead minnow	96-hour LC50, 3.1 mg/l	S	21	pH 7.0–7.4 HD 44
	Fathead minnow				
	Juvenile	96-hour LC50, 6 mg/l	S	21	pH 7.0 HD 150
Zinc[233]	Fathead minnow	270-day MATC, 78–145 μg/l	R	25	pH 7.7 HD 45–47
Zinc[209]	Steelhead trout	96-hour LC50, 1 755 μg/l	F	10.3	pH 7.45 HD 83
	Coho salmon	96-hour LC50, 905 μg/l	F	13.7	pH 7.4 HD 25
Zinc[210]	Rainbow trout				
	Juvenile	96-hour LC50, 0.55 mg/l	F	15	pH 7.4–7.6 HD 44–47
	Juvenile	96-hour LC50, 2.5 mg/l	F	15	pH 7.2–7.4 HD 170–180
	Brook trout (*Salvelinus fontinalis*)				
	Juvenile	96-hour LC50, 2.0 mg/l	F	15	pH 7.4–7.6 HD 44–47
	Juvenile	96-hour LC50, 6.0 mg/l	F	15	pH 7.2–7.4 HD 170–180
Zinc[201]	Chinook salmon				
	Alevin-smolt	96-hour LC50, 97–701 μg/l	F	12.2	pH 7.1–8.3 HD 24
	Steelhead trout				
	Alevin-smolt	96-hour LC50, 93–815 μg/l	F	12.2	
Zinc[234]	Brook trout	96-hour LC50, 2 000 μg/l	F	9	pH 7.2–7.9 HD 45.5
		3-year MATC, 534–1 360 μg/l	F	9	pH 7.0–7.7 HD 45.5

TABLE I—(*Continued*)

Pollutant and Reference Number	Species	Results	Exposure Type	Temperature (°C)	Test Conditions
Zinc[222]	Atlantic salmon Juvenile	21-day LC50, 0.35–1.6 mg/l	F	10	pH 6.1–7.1 HD 12–24
Zinc[211]	*Rasbora daniconius*	96-hour LC50, 38.1 mg/l	R	24.5	pH 7.2–7.7
		240-hour LC50, 33.3 mg/l	R	24.5	HD 230–280

[a] S = static bioassay
[b] HD = hardness, mg/l
[c] F = flow-through bioassay
[d] BCF = bioconcentration factor
[e] MATC = maximum acceptable toxicant concentration

multiple toxicity approach to evaluate the toxicity of chemical combinations.

Takeda and Itazawa [41] and Itazawa and Takeda [42] determined the DO requirement for carp (*Cyprinus carpio*) from measurements of several blood and respiratory parameters. In the first study the minimum level of ambient DO required for the maintenance of normal levels of partial pressure, percentage of saturation, and O_2 concentration in arterial blood was 70–80 mm Hg or 45–55% saturation. However, a lower level of 30 mm Hg was estimated in a subsequent study because it was found that, if expired CO_2 was removed from the water, PCO_2 in arterial blood was reduced and the O_2 affinity of the blood was increased. Simulating drought environments, Cech *et al.*[43] varied temperature and DO levels in flow-through respirometers and measured the respiratory metabolism of largemouth bass (*Micropterus salmoides*). The results showed that the "critical" O_2 tension for this species at 20°C lies below 40 Torr, at 25°C between 40 and 50 Torr, and at 30°C between 50 and 60 Torr.

An examination of O_2 uptake and transport processes by Burggren and Randall [44] clearly identified the sturgeon (*Acipenser transmontanus*) as an O_2 conformer with a critical O_2 tension just below air saturation. Measurements of gill ventilation; stroke volume and frequency; percentage of O_2 utilization and O_2 uptake; and dorsal aortic blood O_2 tension, content, pH, and O_2 affinity during normoxia (150 mm Hg) and during a range of hypoxic exposures (100–30 mm Hg) led to the conclusion that the sturgeon reduces total energy expenditure during hypoxic exposure rather than switching from aerobic to anaerobic metabolism.

Two papers focused on the effects and relationships of several factors to the bioenergetics of the fathead minnow.[45, 46] The effects of weight, temperature, group size, and DO levels on the fish were determined, and equations were presented relating these to O_2 consumption (metabolism). The results showed fathead minnows to be neither true conformers nor regulators, but partial regulators and conformers depending on the O_2 level, with a breakpoint at about 5 cm^3 DO/l. Studies on the effect of pH showed that a curvilinear relationship exists between pH and O_2 consumption and that a maximum metabolic rate occurred at pH 8.7, with little or no O_2 consumption at pH near 5.9 and 11.2. Schulze-Wiehenbrauck [47] measured the O_2 consumption of juvenile cichlids (*Tilapia zillii*) exposed to waters containing water soluble fish excreta (presumably high in ammonia and nitrites). Compared to single fish, which showed a reduced O_2 consumption, grouped fish showed a slight increase in aerobic metabolism and decreased energy utilization, thus confirming other results found in crowded conditions.

Itazawa and Takeda [48] presented data on gas exchange at the gills of carp under normoxic and hypoxic conditions. Compared to other teleosts and elasmobranches in similar conditions, O_2 pressure in dorsal aortic blood during hypoxia was lower, but the higher O_2 affinity of carp hemoglobin maintained nearly full O_2 saturation in arterial blood. The most evident physiological adjustment to hypoxia was increased ventilation frequency, although increased perfusion at the gills played a small role. Two studies investigated blood respiratory properties in the carp.[49, 50] The effects studied were the role played by factors such as pH, enzymes, metal ions, O_2 tension, O_2 transportation, and effects resulting from environmental hypoxia. Blood pH in the armored catfish (*Hypostomus* sp.) induced to breathe

air when held in hypoxic water was observed to be the same as that of fish kept in well-aerated water.[51] Although CO_2 retention occurred in the hypoxic group, indicating reduced aquatic gas exchange, it was fully compensated by increased plasma bicarbonate. Several hematological parameters in facultative air-breathing Amazonian fish after acclimation to normoxic ($PO_2 = 125$ mm) and hypoxic ($PO_2 = 20$ mm) water were measured by Weber *et al.*[52] During hypoxia, armored catfish (*Hypostomus* sp. and *Pterygoplichthys* sp.) intermittently surfaced to gulp air, resulting in lower erythrocyte nucleoside triphosphate concentrations and, subsequently, increased blood O_2 affinity. Similar changes were not seen in the eel (*Synbranchus*), which air-breathes almost continuously when kept in hypoxic water.

Oxygen uptake rates, breathing behavior, and breathing responses, along with blood respiratory properties in two closely related teleosts, the osteoglossids *Arapaima gigas*, an obligatory air breather, and *Osteoglossum bicirrhosum*, an exclusive water breather, were compared.[53] Because of a striking difference in the O_2 affinities of red cell suspensions, it was concluded that the O_2-binding properties of the hemoglobin molecules of the two species are intrinsically different.

The effect of asphyxiation stress on serum glutamic-oxylacetic (SGOT) and serum glutamic-pyruvic transaminase (SGPT) levels in the catfish (*Clarias batrachus*) was studied by Tandon and Chandra.[54] Both enzyme levels fell immediately after sudden asphyxiation stress of 15 minutes, but the SGOT level rose again as the stress continued and was 22.16% higher than the initial level after 60 minutes. SGPT also rose with 15 to 30 minutes stress, but fell again until, at the end of 60 minutes, it showed a loss of 24.54% from the initial level of control fish.

Van den Thillart and Kesbeke[55] measured the production rates and whole fish concentration of CO_2, ammonia, lactate, and glycogen in goldfish (*Carassius auratus*) held at complete anoxia (3% air saturation) for 10 hours. The results showed that glycogen was oxidized into CO_2, suggesting that an unknown electron acceptor must be involved in anaerobic energy production. Blood pH level remained unchanged, but bicarbonates and PCO_2 in blood and tissues of *Cyrpinus carpio* increased with the aquarium water concentration of carbonic acid (HCO_3^- and CO_2) in tests conducted by Zakharenko *et al.*[56] Also elevated in fish with increased blood carbonic acid was the amount of total and residual nitrogen plus several metabolites.

Farmer[57] examined the effects of CO_2 on hemoglobin function in four species of fish, two of which were air breathers, and one amphibian. The carbon dioxide effect of O_2 affinity was similar for all species except for a somewhat larger effect in the Amazonian catfish (*Brachyplatystoma* sp.). The drop in O_2 affinity because of CO_2 increased with increasing pH for each hemoglobin and was about half that produced by CO_2 for human HbA. The dynamics of exchange between inorganic ions of HCO_3^-, Cl^-, K^+, Na^+, Ca^{2+}, and Mg^{2+}, along with their distribution in tissues and excretion in relation to changes in acid-alkaline balance caused by hypercapnia in CO-treated carp were discussed by Romanenko *et al.*[58] Reabsorption of some elements by the kidneys may have been intensified during the chronic experiments. Blood ammonia and urea concentrations in coho salmon exposed to three concentrations of NH_4Cl (3, 16, and 47 mg N/l) or 30% unchlorinated secondary-treated wastewater (3.4 mg N/l) for 91 days were not significantly different regardless of ambient ammonia concentration.[59] However, hemoglobin and hemotocrit decreased; circulating immature erythrocytes increased, as well as plasma Na^+ concentration; and blood glucose decreased as liver glycogen stores increased. Shaffi and Prasad[60] measured the glycogen content in tissues of freshwater catfish (*Clarias batrachus*) in an attempt to study the effect of sodium cyanide on tissue energy reserve. In fish exposed to 1, 3, and 5 mg/l for 2 hours, glycogen decreased at all concentrations in liver, muscle, and kidney tissue but increased in brain tissue at 1 and 3 mg/l. However, glycogen breakdown was again maximal in brain tissue after exposure to 5 mg/l.

The locomotor responses of two cyprinodontid fishes to various DO concentrations were compared by Hill *et al.*[61] *Cyprinodon rubrofluviatilus* showed extreme tolerance and little response to concentrations between 1.5 and 20.0 mg DO/l, whereas *Fundulus zebrinus* spent substantially less time in O_2 concentrations below 4.0 and above 11.0 mg/l than in the control concentration of 6.0 mg/l. In addition, *F. zebrinus* spent more time at 8.0 mg/l than in all other concentrations, suggesting a relatively narrow optimal O_2 concentration for this species. As DO declined from normoxic ($PwO_2 \geq 150$ Torr) to hypoxic ($PwO_2 < 50$

Torr), almost all of the 26 species of great plains fishes tested by Gee *et al.*[62] were able to use DO in surface water even though these species are not well adapted to use this O_2 source indefinitely. Only the *Salmonidae* (three species) and *Stizostedion vitreum* did not move to the surface and breathe the surface film. Because all the fishes showed similar sequential behavior patterns during progressive hypoxia, certain points in this sequence were used to estimate incipient limiting and lethal levels of DO. Air breathing catfish (*Heteropneustes fossilis*) subjected to fluctuations in PO_2 and CO_2 surfaced more frequently and swam longer distances as PO_2 dropped and CO_2 inversely increased, compared with individuals maintained in aerated water (152 mm Hg).[63] Although food intake increased with decreasing PO_2, the conversion rate and efficiency of these fish decreased as the fish spent more energy on metabolic processes, including excretion, and the fish, consequently, showed poor growth.

Smith and Jones [64] located receptors causing bradycardia in trout (*Salmo gairdneri*), identified their innervation, and discussed the biological advantage of hypoxia bradycardia in fish. The secondary lamellae of gills from hypoxia and anesthetized rainbow trout were analyzed by Soivio and Hughes [65] for blood volume and hemotocrit value (Hct). The gills underwent vasodilation combined with a slight decrease in Hct in hypoxia, vasoconstriction but continuing decrease in Hct during recovery from hypoxia, vasodilation combined with increased Hct in anesthesia, and continuing vasodilation but falling Hct to below that of controls during recovery from anesthesia. Hughes *et al.*[66] carried out morphometric analyses of different portions of gills from rainbow trout kept under normoxia or hypoxia. Although there were some overall differences in the blood components of filaments from dorsal, lateral, and ventral regions as well as parts of a filament, these were insignificant compared to the differences observed between corresponding material fixed in either 2.5% glutaraldehyde or 2.5% glutaraldehyde + 2.5% formaldehyde. The differences were greater during normoxia than during hypoxia.

Erythropoietic regulation in the Florida gar (*Lepisosteus platyrhincus*) was examined by McLeod *et al.*[67] Exposure to hypoxia (one-third of normal O_2 tension), CO, or drugs known to stimulate mammalian erythropoiesis did not stimulate red blood cell production, nor did alteration of plasma volume without decreasing cell mass. The gar did respond to bleeding by increased erythropoiesis, and the hypothesis was advanced that hypoxia failed to stimulate erythropoiesis because erythropoiesis in these fish is regulated by cell mass feedback (cell-to-cell interaction) and is independent of O_2.

The prevalence and severity of nephrocalcinosis, characterized by calcification and granuloma formation in kidney and stomach, increased with increasing environmental free CO_2 concentration in rainbow trout exposed a minimum of 275 days to CO_2 concentrations ranging from 12 to 60 mg/l.[68] The diet, but not water, PO_4-P concentration modified the prevalence and severity at each CO_2 concentration tested. Bohl [69] found that hyperemia, swelling of the gill system, mucus secretion, developing whitish-grey layers on the gills, partial respiration, and whirling is symptomatic of gill necrosis in carp. The disease, supposedly a pH-dependent autintoxication by ammonia, can be prophylactically controlled by adjusting the pH and ammonium value with 15 kg chlorinated Ca/ha, three to five times every second day. Murachi *et al.*[70] proposed that measurement of the blood corpuscle resistance (BCR) of fish within several hours after death may indicate whether or not death was caused by environmental hypoxia. BCR in carp significantly decreased immediately after death from environmental hypoxia but remained normal when the fish died of cyanide poisoning. However, compared to these levels, the BCR increased in the environmental hypoxia case at 24 hours post mortem, whereas it decreased in the cyanide poisoning case.

pH. Numerous lakes in Maine decreased in pH from an average of 6.85 in 1937 to an average of 5.95 in 1974, representing an 8-fold increase in acidity.[71] These results were correlated with a history of acid precipitation. No biological effects were discerned because the decrease in pH was moderate compared to decreases in other areas, where more severe acidification has resulted in severe effects on fish and other biota. Letterman and Mitsch [72] studied the effects of acid mine drainage on the fish and macroinvertebrate populations of an otherwise unpolluted stream in western Pennsylvania. The pH remained between 6.5 and 8 below the mine discharges because of alkaline discharge and upstream alkalinity. Deposition of ferric hydroxide ranged from 3 $g/m^2 \cdot d$ near the discharges to 0.01 $g/m^2 \cdot d$

at the furthest downstream study site. The standing crop of fish decreased from 228 to 11 kg/ha, with the benthic fishes such as sculpin (*Cottus bairdi*), white sucker (*Catostomus commersoni*), and hog sucker (*Hypentelium nigricans*) being the groups most affected. The effect of acid mine drainage on fish populations in the upper Potomac River and its associated tributaries was discussed by Davis.[73] A sampling program for this area showed no fish at the three uppermost stations on the North Branch of the Potomac River, nor from 14 tributary stations, because of heavy acid mine drainage. Only 3 to 9 species and sparse populations were found in those streams receiving intermittent to small amounts of acid mine drainage, whereas streams with no acid mine drainage had from 3 to 24 species in abundance. Water quality was sufficiently improved further down the Potomac to allow establishment of 22 species.

Hatchery-reared brook trout (*Salvelinus fontinalis*) exposed to water of low pH showed pronounced strain differences in survival ability among embryonic, juvenile, and adult trout in laboratory tests with H_2SO_4 solutions, and among juvenile trout in field tests with mine acid polluted waters.[74] Of the factors examined in hatchery trout for enhancement of acid resistance, acclimation of the fish to stream or laboratory conditions of controlled pH before acid exposure and the hereditary resistance of the given strain were most important. Over a 3-year period, Carrick [75] investigated the effect of acid water on the hatching of salmonid eggs. Although there was considerable yearly variation in hatchability, there were no apparent differences in the tolerance of eggs from salmon (*Salmo salar*), sea trout, and brown trout (*Salmo trutta*) to acid water in the pH range 3.5–7.0. A pH of 3.5 was lethal within 10 days to all the eggs, but at pH 4.5 and higher there was no obvious difference in hatching attributable to acidity. Witschi and Ziebell [76] advised against stocking hatchery-reared trout accustomed to nearly neutral pH water into lakes with a pH of 9.0 or higher. For rainbow trout (*Salmo gairdneri*) transferred from water with pH 7.2 into water with pH ranging from 8.5 to 10.0 for 48 hours, survival was 88% at pH 9.0, 68% at pH 9.5, and 0% at pH 10.0. After 48-hour exposure, only a few survivors in pH 9.0 and none in pH 9.5 fed when offered their usual pelleted food. In addition, pH-stressed fish were sluggish and suffered loss of equilibrium, making them vulnerable to predation.

Sulfuric acid was evaluated as a control for nuisance weeds by Trent *et al.*[77] Results showed that while H_2SO_4 at pH 3 produced no toxic effects on hydrilla (*Hydrilla verticillata*), it was extremely toxic to all faunal groups tested. Of the vertebrates, mosquitofish (*Gambusia affinis*) were more tolerant to pH changes than largemouth bass (*Micropterus salmoides*), but mortality at pH 3 was 80% after 24 hours. Rainbow trout developed acidemia, which was not the result of anaerobic respiration when exposed to environmental acidification.[78] After 5 days at pH 4.0 and normocapnic conditions, dorsal aortic blood samples showed a significant decrease in pH and total CO_2 but none in PO_2 and lactate compared to controls. After 12 days there were significant increases in hemoglobin, hematocrit, and erythrocyte levels. Brook trout exposed to an acidic environment (pH 3.2–3.5) developed severe metabolic acidosis, as shown by decreased plasma bicarbonate levels, decreased blood pH, and negative base excess values.[79] In acidotic trout, reduced O_2 consumption resulted from decreased O_2 transfer across the gills and reduced available blood O_2 capacity.

Haranath *et al.*[80] studied the effect of altered pH on protein metabolism in *Tilapia mossambica*. Compared to controls at pH 7.2, fish exposed to pH 6 or 9 for 24 hours showed increased protease activity in liver tissue. Muscle tissue was different in that it accumulated proteins, either from active protein synthesis or reduced proteolysis. Free ammonia increased dramatically in liver tissue at pH 9, while urea increased considerably at pH 6, suggesting that formation of nitrogenous end products by the liver may depend on media pH. Blood pH in medaka (*Oryzias latipes*) exposed to pH 4.0 for 4 hours dropped from 7.34 to 6.99, and plasma Na concentration decreased from 110.5 to 66.4 mEq/l.[81] The plasma Na level was maintained at the normal level when Ca ion was present at a concentration of 30 m*M* and, subsequently, survival time of the fish in an acidic environment was increased. Wood and Caldwell [82] provided first evidence that the freshwater fish kidney dynamically regulates acid-base status. Intra-arterial injection of a fixed acid load caused only a short-lived (<2-hour) disturbance of blood pH but a long-lived (2- to 3-day) elevation in urinary acid excretion in trout (*Salmo gairdneri*). The total elevation in urinary acid efflux over 72 hours was such that renal function was the only mechanism needed to

explain compensation of this experimental "metabolic acidosis."

CHEMICAL POLLUTANTS

Insecticides. In order to evaluate the role of DDT and PCBs on reproductive failure of lake trout (*Salvelinus namaycush*) in Lake Michigan, Stauffer [83] studied the mortality of eggs and fry from planted and hatchery-reared fish. Mortality of eggs and fish from Lake Michigan in 1973–1974 was greater than the mortality of hatchery eggs and fry, but mortality of eggs and fry in 1974–1975 from both sources was similar. Mortality of eggs and fry of Lake Michigan fish during different stages of development was not linearly correlated with DDT or PCB content of eggs. The bulk of the evidence indicated that DDT and PCBs did not have an effect on early survival of lake trout from Lake Michigan.

Bulkley [84] compared the variation in concentration of DDT in muscle tissues of channel catfish (*Ictalurus punctatus*) from the Des Moines River in Iowa. Residue concentrations varied widely but were significantly correlated with body length, age, and fat content. Residue concentrations were not correlated with the sex of the fish. The distribution of chlorinated hydrocarbon pesticide residues was measured in Lake Monroe, Ind., from June 1969 to June 1971.[85] Water concentrations of aldrin, dieldrin, and lindane were usually <10 μg/l, with a maximum concentration of 4 μg/l for aldrin and dieldrin. Most fish contained only trace or nondetectable levels, but some exceeded 50 ng/g. Pesticide levels in fish were variable and were not correlated with length or weight. Perry [86] monitored pesticide and PCB residues in the Upper Snake River following the collapse of the Teton Dam in 1976. Thirteen toxicants were identified. Rainbow trout (*Salmo gairdneri*), Utah sucker (*Catostomus ardens*), and mountain whitefish (*Prosopium williamsoni*) contained a maximum of 1 432, 1 420, and 2 650 ng/g total DDT and analogs; 66, 32, and 30 ng/g dieldrin; and 1 010, 1 800, and 1 400 ng-g PCBs, respectively. Organophosphates were not detected in the fish samples. Heptachlor epoxide; BHC; lindane; 2,4-D; and carbaryl residues were detected in some fish samples. Organochlorine pesticides and PCB residues in one specimen of sea lamprey (*Petromyzon marinus*) from Lake Ontario, Canada, were measured by Kaiser and Valdmanis.[87] Eleven organochlorine pesticide contaminants were detected at concentrations similar to those found in salmonid species. The concentration of DDT and metabolites and mirex exceeded the existing guidelines and proposed water quality objectives.

The behavior and persistence of some pesticides in aquatic systems were investigated by Sharom.[88] The results of these studies were used to explain the persistence and fate of pesticides found in the Holland Marsh area, Ontario, Canada. Insecticide residues in water, sediment, and fish of the drainage system of Holland Marsh in 1972–1975 were measured by Miles and Harris.[89] In the Holland Marsh drainage ditch, total DDT concentrations exceeded 5 μg/g in carp (*Cyprinus carpio*) and catfish with p,p′-DDE comprising 42% and p,p′-TDE comprising 39% of the total DDT. Dieldrin residues varied from 0.16 to 0.95 μg/g in the various groups of fishes. Diazinon, ethion, α-endosulfan, and γ-chlordane at concentrations less than 0.1 μg/g were detected in some groups of fishes. Residue levels of fish from Lake Simcoe, into which the Holland Marsh drains, were limited almost completely to DDT. The percentage of p,p′-DDT in these fish was much greater than in the fish from the drainage ditch. In another study, mean residues of total DDT in Lake Simcoe in 1970 ranged from 0.33 μg/g in burbot (*Lota lota*) in 14.74 μg/g in large lake trout (*Salvelinus namaycush*).[90] By 1975–1976 the mean concentration of total DDT declined significantly ($P = 0.05$) in all species except small walleye (*Stizostedion vitreum vitreum*) and large lake trout. The mean residues of dieldrin in 1970 ranged from 4 ng/g in burbot to 205 ng/g in larger lake trout. By 1975–1976 residues had declined significantly in five species, showed no change in two, and in two species the larger fish had lower residues and the smaller fish showed no change. Other organochlorine insecticides detected were chlordane, heptachlor, and hexachlorobenzene.

Terpenes and chlordane components in fish, guillemot, and seal from Swedish water were identified by Jansson *et al.*[91] Fish samples from the Baltic Sea, North Sea, and Lake Vättern were found to be contaminated with chlorinated terpenes. Chlordane-related compounds were also found in all samples, with trans-nonochlor and oxychlordane being more common.

Gajdúšková and Maytas [92] measured residues of pesticides in pond water and carp from the territory of the Pohorelice State Fisheries En-

terprise, Czechoslovakia. Organophosphorus pesticides were not detected in any of the samples. The gamma isomer of HCH was the only pesticide found above trace levels in water. The following mean and maximum values of pesticides were found in carp muscle (μg/g): HCB, 0.172 and 0.434; αHCH, 0.160 and 0.440; γHCH, 0.155 and 0.434; DDE, 0.357 and 0.900; and DDT, 0.383 and 0.800. The residues of some chlorinated hydrocarbon pesticides in the hare, pheasant, and carp from Czechoslovakia were measured by Kacmar *et al.*[93] In the liver of carp, DDT levels ranged from 0.18 to 0.56 μg/g and HCH levels ranged from 0.23 to 1.0 μg/g. Levels in the muscle varied from 0.33 to 0.48 μg/g of DDT and 0.037 to 0.1 μg/g of HCH.

Lipka and Doboszynska[94] measured DDT residues in 11 species of freshwater fish caught in 1972–1974 in northern Poland. DDT levels in these fish ranged from 0.05 to 4.0 μg/g. Chlorinated pesticide residues in fish from two Teheran, Iran, water supply reservoirs were measured by Hashemy-Tonkabony and Gharibzadeh.[95] Each sample was screened for nine or more chlorinated pesticides, but DDT and its metabolites DDE and TDE were the principal pesticides found, with DDE being found in all species of fish. Residues of DDT were found in the muscles and livers of six species of fish from Lake Nubia, Sudan.[96] Only 10 of 58 fish contained detectable levels. All 10 samples contained p,p′-DDE, while p,p′-DDT was found only in three muscle samples. The highest level of total residue, 184 μg/kg, was found in a muscle sample of *Hydrocynus forskalii*. Greichus *et al.*[97] measured insecticides, PCBs, and metals in the water, sediments, and aquatic life of Lake Nakuru (The Lake of a Million Flamingos), Kenya. The fish, *Tilapia grahami*, had residue levels of 0.02, 0.02, 0.01, and <0.01 μg/g of DDE, dieldrin, DDD, and DDT, respectively.

The effects of experimental contamination using endrin and caged and free-living aquatic animals in a pool was studied by Wohlgemuth and Trnkova.[98] The pool (10 × 10 m with an average depth of 0.44 m) was treated at one corner with endrin (20% EC) at a dose calculated to give a concentration of 2.5 mg/l. All caged rainbow trout and goby (*Gobio gobio*) died within 24 hours. In general, the tadpoles and aquatic invertebrates were more resistant than the fish.

The effects on stream fishes and invertebrates of an operational spraying of acephate to suppress spruce budworm was investigated by Rabeni and Stanley.[99] Acephate reached its maximum concentration of 140 μg/l in North Brook and 113 μg/l in South Brook within an hour after spraying with residue remaining for at least 2 days. Brain AChE activity of caged fish was depressed in the longnose sucker (*Catostomus catostomus*) but not in the brook trout (*Salvelinus fontinalis*) or Atlantic salmon (*Salmo salar*). Brook trout captured 1 and 2 days after the spraying had consumed significantly more terrestrial invertebrates than before treatment, but the effect on trout diet was transient. The growth of brook trout and Atlantic salmon was about the same between the two treated streams and the control stream.

Herman and Bulger[100] studied the effects on nontarget organisms of a forest spraying of DDT to control an outbreak of Douglas-fir tussock moth. The mean of total DDT residues in rainbow trout before spraying was 0.11 μg/g, and after spraying the mean was 0.60 μg/g with a maximum of 2.6 μg/g. About half of the total DDT was p,p′-DDT. The mean total DDT residues in sculpins (*Cottidae*) before spraying was 0.01 μg/g. After spraying the mean was 0.32 μg/g and 0.54 μg/g in free-swimming and caged fish, respectively. About 70% of the DDT was p,p′-DDT. Suckers (*Catostomidae*) showed less than an order of magnitude increase, mostly DDT, following the spray operation.

The toxicity of some insecticides to the loach (*Lepidocephalus thermalis*) was investigated by Rita-Kumari and Balakrishnan-Nair.[101] The reported 48-hour LC_{50} values for demicron, malathion, metacid, parathion, and endrin are shown in Table I. Smaller fish were found to be more sensitive to these insecticides than larger ones. The acute toxicity of aldrin and ethyl parathion was reported for patra (*Notopterus notopterus*) and kharda (*Colisa fasciatus*).[102] Under all experimental conditions, the 96-hour LC_{50} value for aldrin was lower than the corresponding value for ethyl parathion, and the 96-hour LC_{50} value for patra was lower than the value for kharda. The 96-hour LC_{50} values for these species are listed in Table I. Statistical analysis of the data indicated that temperature, DO, pH, and size of fish had a significant effect on the LC_{50} values for both compounds and both species of fish. Puiseux-Dao *et al.*[103] determined the acute LC_{50} values of some pesticides (Table I) and detergents using three species of unicellular algae, two species of aquatic invertebrates, two species

of amphibians, and trout (*Salmo irideus*) fry. Also the effects of some pesticides at the cellular level were investigated.

Coats and O'Donnell-Jefferey [104] reported on the acute toxicity of four synthetic pyrethroid insecticides, both technical and formulated grades, to rainbow trout. The 24-hour LC_{50} values of the technical-grade material ranged from 55 μg/l for cypermethion to 135 μg/l for permethrin. In all cases the formulated material was more toxic in terms of active ingredients with 24-hour LC_{50} values ranging from 8.6 μg/l for fenpropanate to 61 μg/l for permethrin. There was no clear trend in the relationship of water solubility and partition coefficients to toxicity. The acute toxicity of permethrin, decamethrin, and three related pyrethroids to the Atlantic salmon and lobster was determined by Zitko *et al.*[105] The 96-hour lethal threshold concentrations varied from 0.59 μg/l for DECIS to 8.8 μg/l for permethrin. The relationship of the octanol/water partition coefficient to toxicity did not apply because the toxicity of the cyano-substituted pyrethroids, decamethrin and NRDC 1685, was greater than predicted and the pyrethroids containing 3-demethylvinyl chrysanthemic acid moiety were less lethal than predicted. The acute toxicity of permethrin to five species of aquatic animals was compared.[106] Crayfish were the most sensitive, and bullfrog tadpoles were the most resistant. The 96-hour LC_{50} values for the three fish species ranged from 1.10 μg/l for the channel catfish to 15 μg/l for the mosquitofish (*Gambusia affinis*). In another study Jolley *et al.*[107] determined the acute toxicity of Pounce, a synthetic pyrethroid, to crayfish, bullfrog tadpoles, and three species of fishes. The 96-hour LC_{50} values for channel catfish, largemouth bass (*Micropterus salmoides*), and mosquitofish are shown in Table I.

Toxicity tests to determine the effectiveness of Ni_2B-catalyzed dechlorination in reducing the acute toxicity of DDT, chlordane, and lindane to bluegill (*Lepomis macrochirus*) and *Daphnia magna* were conducted by Randall *et al.*[108] The 96-hour LC_{50} value of chlorinated technical-grade material for the bluegill was over 1 000 times greater for DDT and lindane and 14 times greater for chlordane. The reduction in toxicity of formulations of these insecticides was less than the reduction in toxicity of the technical-grade material. The 96-hour LC_{50} value of the dechlorinated lindane reaction mixture was closer to those of the Ni_2B-catalyzed dechlorination system itself than were the 96-hour LC_{50} values of a DDT and chlordane mixture.

The chemistry and aquatic toxicity of the degradation of diazinon by sodium hypochlorite showed that diazinon was rapidly degraded by this compound between pH 8 and 10.[109] Treatment with hypochlorite reduced the acute toxicity of diazinon dust formulation to bluegill by at least 3 700-fold. The reduction in toxicity for the fathead minnow (*Pimephales promelas*) was less.

Billard [110] determined the effects of lindane, metals, and temperature on fish reproduction. Lindane had no effect on gametogenesis in rainbow trout, but it caused a decrease in fertilization at a concentration of 25 mg/l. Mercury, Fe, Cu, and Cr were the most toxic metals for gametes.

Curtis and Beyers [111] examined the effect of kepone exposure on the reproductive capacity of female medaka (*Oryzias latipes*). Female fish were exposed to kepone for 12 hours a day and then returned to breeding tanks containing males. After 94 hours of exposure to 2 μg/l, no fish continued to lay eggs, and after a 108-hour exposure, 30% of the females died. There was no significant difference in mean egg weight or clutch size for any group of fish.

The effects of dieldrin on the growth and body composition of rainbow trout fed Oregon moist pellets or turbificid worms were investigated and the results showed that at a feeding rate of 2.5, 4.0, and 5.5% of body weight per day, trout fed tubificid worms grew faster than fish fed Oregon moist pellets.[112] Flow-through exposure of fish to 0.18 μg/l of dieldrin for 61 days did not affect protein elaboration of trout fed either diet or fat metabolism in fish fed Oregon moist pellets. However, dieldrin exposure did reduce fat elaboration in trout fed tubificids. The dieldrin concentration and percent of fat increased as feeding levels increased.

Jarvinen and Tyo [113] determined the chronic toxicity and residues of endrin in fathead minnows exposed to endrin in the water or food, or both. The food was clams that had accumulated ^{14}C-endrin when exposed to a water concentration of endrin similar to that to which the fish were exposed. Higher endrin residues were accumulated from water than from the food, with concentration factors of 13 000 and 0.8 from these two sources, respectively. Residues contributed by endrin in the food (0.63 μg endrin/g food) were additive to those from the water at all life history stages. Survival of

30-day progeny was significantly reduced at a water concentration of 0.14 μg/l.

Isensee *et al.*[114] determined the acute toxicity to bluegill of toxaphene and nine of its fractions (Table I) and measured the fate of toxaphene and three of its fractions (^{14}C-labeled) in a recirculating static model ecosystem. The bioconcentration factor for bluegills exposed to the higher concentration was 8 100 for toxaphene, 4 300 for Fraction 5, 4 200 for Fraction 9, and 840 for Fraction 1. Thin-layer chromatographic analysis of fish extracts indicated little metabolism of toxaphene or the three fractions. The absorption and elimination of ^{14}C-*cis*-chlordane and ^{14}C-photo-*cis*-chlordane by goldfish (*Carassius auratus*) was compared by Ducat and Kahn.[115] After a 16-hour exposure to 10 μg/l, the concentrations of *cis*-chlordane and photo-*cis*-chlordane in the fish were 2.96 and 3.52 mg/kg, respectively. Fish exposed to 25 μg/l for 4 days had the highest concentrations of both compounds in the bile. The half-life of these two compounds in fish exposed to 5 μg/l and returned to clean water was 7–10 days for photo-*cis*-chlordane and about 5 weeks for *cis*-chlordane. The bioconcentration of diazinon by fish, snails, and crayfish in a flow-through exposure was investigated by Kanazawa.[116] The BCF of diazinon by fish was generally greater than that of crayfish or snails. The BCF determined from a 7-day exposure to 10 μg/l in a flow-through system was 152 for topmouth gudgeon (*Pseudorasbora parva*), 65 for carp, 37 for silver crucian carp (*Cyprinus auratus*), and 18 for guppies (*Labistes reticulatus*). The BCF for diazinon by topmouth gudgeon reached equilibrium after a 3-day exposure to 10 μg/l and a 4-day exposure to 50 μg/l. The depuration rate was rapid with over 99% being lost in 7–8 days.

Matthiessen and Johnson[117] described the uptake and fate of Abate in the tropical food fish *Sarotherodon mossambicus* exposed to concentrations similar to those produced in West Africa rivers by the WHO Onchocerciasis Control Programme. The fish absorbed Abate directly from the water and from contaminated food. Internal distribution depended on tissue fat content. Fed fish exposed to a flow-through concentration of 0.05 mg/l contained 2.3 μg/g after 29 days and 8.86 μg/g after 69 days, while starved fish contained 14.55 μg/g after 29 days and 16.85 μg/g after 69 days.

The accumulation and elimination of diflubenzuron by bluegills and white crappie (*Pomoxis annularis*) showed that both species accumulate diflubenzuron up to 80 times when exposed to 10 μg/l for 24 hours in a static test.[118] When returned to clean water, exposed bluegills eliminated diflubenzuron within 48 hours, and neither the parent compound nor the primary hydrolytic product, p-chlorophenylurea, were important excretary products.

Sastry and Sharma[119] examined the effects of 0.03 mg/l of endrin for 30 days on acid phosphatase, alkaline phosphatase, and glucose-6-phosphatase in the liver and kidney of *Channa punctatus*. There was a significant increase in the activity of alkaline phosphatase in the liver after 15 and 30 days, an increase in the glucose-6-phosphatase in the liver after 30 days, and an increase in the glucose-6-phosphatase in the kidney after 15 and 30 days. The effects of enzyme activity in the liver and kidney of *Channa punctatus* exposed to 0.01 mg/l endrin for 30 days was reported by Sharma and Sastry.[120] A significant effect was found on the activity of acid phosphatase and α-amylase in the liver and on the activity of glucose-6-phosphatase and lipase in the kidney. Two other studies determined the effects of endrin on the enzyme activity in the liver and kidney of *Ophiocephalus punctatus* exposed to this compound for 20 days.[121, 122] After exposure to 0.01 mg/l, there was a significant increase in the acid phosphatase and glucose-6-phosphatase in the kidney. The activity of alkaline phosphatase in the liver was significantly affected at 0.03 mg/l. The *in vitro* effects of aldrin and dieldrin on total ATPase; Mg^{2+} ATPase; and Na^+, K^+ ATPase of tissue homogenates of brain, gill, liver, and kidney of *Labeo rohita* were measured by Verma *et al.*[123] For aldrin a concentration of 1.66 μ*M* significantly lowered the activity of Na^+, K^+ ATPase of the gill. Dieldrin at a concentration of 1.66 μ*M* significantly lowered the activity of all three ATPases of the kidney, total ATPase of liver, and total ATPase and Mg^{2+} ATPase of gill. At a concentration of 0.55 μ*M*, dieldrin significantly lowered all three ATPase activities in the brain. At the low concentration of 0.06 μ*M* for both compounds, stimulation was found in all tissues except gill Mg^{2+} ATPase activity.

Another study determined the effects of chlordane upon the ATPase system in the brain, gill, liver, and kidney of roho (*Labeo rohita*) and singii (*Saccobranchus fossilis*).[124] Fish were exposed for 30 and 60 days to 1/2, 1/3, and 1/6 of the 96-hour LC_{50} concentration in a static, renewal system. There was a sig-

nificant inhibition of all three ATPases in all four tissues following both 30- and 60-day exposure to the high concentration of 0.09 mg/l for roho and 0.21 mg/l for singii. At the intermediate concentration the percentage of inhibition was less with the activity of some ATPases not significantly lowered.

The effects of endosulfan on the ATPase activity in brain, gills, liver, and kidney of *Channa gachua* exposed in a static, renewal test for 30 and 60 days were reported by Dalela *et al.*[125] The results showed that inhibition was greatest in oligomycin-insensitive Mg^{2+} ATPase in brain, gills, and liver tissue. At the low test concentration of 1.74 μg/l (1/6th of the 96-hour LC_{50}) there was no effect on the ATPase activity of the kidney or brain, but there was an effect at 2.13 μg/l (1/5th of the 96-hour LC_{50}). Some fractions of ATPase activity of gills and liver were significantly greater at 1.74 μg/l.

Kapur *et al.*[126] measured the effect of fenitrothion on the 3 β-HSD of gonads of carp. Fish were exposed to 1.5 and 0.30 mg/l of fenitrothion in a static test, renewed every 7 days for 6 months. For both male and female fish, the difference in the activity of this enzyme between exposed fish and control and between the two exposed groups was significant ($P < 0.01$). In the high concentration the activity of 3 β-HSD was reduced 29% in the male and 16% in the female.

Chin and Sudderuddin[127] determined the 96-hour LC_{50} values of lindane, carbaryl, diquat, and methamidophos to carp (Table I) and studied the effect of methamidophos on growth and the esterase activity of the brain and liver of carp. Enzyme inhibition increased with increasing concentrations and time of exposure. Brain carbarylesterase (CarE) was more sensitive than acetylcholinesterase (AChE), but the reverse was true for the liver. In fish exposed to 20 mg/l of methamidophos for 48 hours and returned to clean water, recovery was nearly complete in 7 days. Liver esterases tended to recover faster than brain esterases, and CarE recovered faster than AChE.

Verma *et al.*[128] measured the effect of exposure to sublethal concentrations of Zolong, Rogor, and malathion on the activity of AChE of the brain, liver, and muscle of *Channa gachua* and *Cirrhina mrigala*. There was a significant decrease in enzyme activity in all tissues of both fish species exposed to the pesticides with increased inhibition with higher concentrations of the organophosphate insecticides.

The inhibition of AChE activity in tissues of Mozambique mouthbrooder (*Sarotherodon mossambica*) exposed to the 48-hour LC_{50} concentration of sevin and sumithion was reported by Koundinya and Ramamurthi.[129] The reduction of AChE activity in all tissues (brain, gill, muscle, liver, intestine, and kidney) of fish exposed to 10 mg/l of sevin or 6 mg/l of sumithion was significant. There was a significant increase in the AChE content of all tissues of fish exposed to these two pesticides. The increase was greater in fish exposed to sumithion. Hematological studies of Mozambique mouthbrooder exposed to 48-hour LC_{50} concentrations of sumithion and sevin showed that both pesticides caused a significant decrease of RBC, packed cell volume, and hemoglobin concentrations.[130] There was no effect on mean corpuscular volume or mean corpuscular hemoglobin concentration.

The effects of a sublethal concentration of methyl parathion on some organic constituents and oxidative enzymes of the muscle, gill, and liver of the Mozambique mouthbrooder were measured by Rao and Rao.[131] Fish were exposed to a static test to 0.1 mg/l of methyl parathion for 48 hours. There was a significant effect on all six parameters of the three tissues, with a decrease in levels of succinate dehydrogenase, lactate dehydrogenase, carbohydrates, and glycogen and an increase in the levels of total free amino acid and total protein.

Shaffi[132] investigated the comparative effects of exposure to 1, 3, and 5 mg/l of heptachlor for 3 hours on liver, muscle, brain, and kidney tissue reserves, serum glucose, and lactate levels in nine food fishes. The decrease of tissue glycogen was greatest in carp, followed by snake-headed fishes, and less in catfish. Increasing concentrations of heptachlor caused greater decreases in liver, muscle, and brain glycogen content. Tissue glycogenolysis from heptachlor exposure was indicated by increased levels of serum glucose and lactate.

The protective role of ascorbic acid on the toxic effects of aldrin on an air-breathing fish (*Channa punctatus*) was reported by Agrawal *et al.*[133] There was 25% mortality of the fish fed a 0.25% aldrin diet, but only 2.5% mortality of fish fed a mixed diet containing aldrin and 0.5% ascorbic acid. Fish fed the aldrin diet had a significant increase in erythrocyte

number, hemoglobin, and hematocrit values as compared to control fish. The fish fed the mixed diet had significantly lower values in erythrocyte number, hemoglobin, and hematocrit values compared to fish fed the aldrin diet.

The effects of diets contaminated with mirex and PCBs on several physiological parameters of coho salmon (*Oncorhynchus kisutch*) and rainbow trout were evaluated in a series of reports.[134-138] The results generally showed that mirex diets (~5–500 μg/g) reduced serum levels, decreased body weight, and decreased carcass lipid content while PCB diets (~50–500 μg/g) reduced serum levels, increased the liver lipid index, and increased liver weight in these fish. Hendricks *et al.*[137] studied the effect of dietary dieldrin on aflatoxin B_1 (AFB_1) carcinogenesis in rainbow trout. Growth reduction in 6-week old fish was significant with both the AFB_1 diet (6 μg/kg) and the dieldrin diet (5 μg/kg) after 12 months. Growth of the fish on the diet containing both compounds was similar to the growth of the fish fed the dieldrin diet. The liver weight/body weight ratios of both groups of fish receiving AFB_1 had a significantly lower ratio than controls.

Plack *et al.*[139] studied the distribution of DDT between the lipoproteins of serum of rainbow trout. ^{14}C-DDT added to the serum either in alcohol or attached to one of the serum lipoproteins or egg lipoproteins was rapidly distributed among all the serum lipoproteins. The concentration of DDT in terms of μg/g lipoprotein lipid varied with the lipoprotein classes but were similar in proportion in all experiments, being in the ratios VLDV: LDL:HDL, 100:76.2:46.0. The effects of intraperitoneally injected DDT and parathion on the concentrations of norepinephrine (NE), 5-hydroxytryptamine (5-HT), and dopamine (DA) in the brain and retina/pigment epithelium/choroid of the goldfish were compared by McDonald[139] and McDonald and Fingerman.[140] DDT elevated the eye tissue 5-HT and decreased the brain NE and DA while it elevated the eye tissue 5-HT, decreased the DA, and had no effect on NE. Parathion lowered brain 5-HT and NE but did not change DA while NE was decreased in the retina/pigment epithelium/choroid but 5-HT and DA were not changed. Both DDT and parathion produced disoriented behavior in the fish. The effects of selected organophosphorus and carbamate insecticides on trout (*Salmo gairdneri*) was investigated by Groba and Trzcinska.[141] No influence of 3.2 mg/l of carbaryl or 5.6 mg/l of propoksur on blood hemoglobin or glucose was found. Lethal and sublethal concentrations of carbaryl and chlorfenwinfos inhibited brain AChE activity. It was concluded that fish brain AChE activity may be a useful tool to evaluate the hazard of organophosphorus and carbamate insecticides.

The effects of sublethal concentrations of five organophosphorus insecticides on temperature tolerance, reflexes, and orientation of mosquitofish were studied by Johnson.[142] The fish were exposed in static tests to ABATE^R, fenthion, chlorpyrifos, methyl parathion, and malathion at concentrations of 1, 5, and 10 μg/l for 24 hours and at a concentration of 5 μ/l for 48 hours. The thermal tolerance was lowered significantly for male and female fish exposed to 5 and 10 μg/l of methyl parathion and chlorpyrifos after 24 hours. Activity was restricted with fish exposed to methyl parathion and chlorpyrifos at a concentration of 10 μg/l for 24 hours and 5 μg/l for 48 hours.

The fate of ^{14}C-*cis*-chlordane in goldfish was determined by Feroz and Khan.[143] Fish were exposed to 26 μg/l of ^{14}C-*cis*-chlordane for 24 hours. Over 99% of the radioactivity recovered from the fish on Day 10 and Day 25 posttreatment was unchanged *cis*-chlordane. The effects of dichlorvos (DDVP) on the respiratory rate and O_2 consumption of three size groups of the mouthbrooder was studied by Rath and Misra.[144] Ventilation rates were positively correlated with O_2 consumption in all age groups. A negative correlation was found between concentration from 0.5 to 1.0 mg/l and O_2 consumption in all size groups of fish exposed for 15 days. In fish exposed to 1.0 mg/l for 21 days, the rate of O_2 consumption decreased with increasing exposure periods.

The effects of endrin on histopathological changes and α-amylase activity of the liver of *Channa punctatus* was determined by Sastry and Sharma.[145] The liver of fish exposed to the LC_{50} concentration of 0.044 mg/l of endrin was characterized by swollen hepatic cells, liver cord disarray, necrosis, and vacuolation of cytoplasm. In addition, the α-amylase activity was reduced by 50%. The effects of intraperitoneal injection of 2.5 μg endrin/g on the histopathological changes in the liver of *Channa punctatus* was described by Sastry and Sharma.[146] Pathological changes became

more severe with an increase of time of exposure from 8 to 24 hours. After a 24-hour exposure there was a marked hypertrophy of the hepatic cells; necrosis was more severe; and the cytoplasm of the cells disintegrated.

The effects on the digestive system of *Channa punctatus* exposed to 0.32 mg/l of Dimecron for 20 days was studied by Sastry and Malik.[147] Pathological changes in the liver were vacuolation of the cytoplasm of hepatocytes, enlargement of nuclei, rupture of the cell membrane, liver cord disarray, damage of connective tissue, infiltration of phagocytes, and necrosis. In addition, there was a significant effect on the activity of alkaline phosphatase of the liver and intestine and on the glucose-6-phosphatase of the liver, intestine, and pyloric caeca. No significant effects on the activity of the acid phosphatase of the liver, stomach, intestine, or pyloric caeca was observed.

Anees[148] described the histopathological effects in the liver of *Channa punctatus* exposed to three levels of three organophosphate insecticides for 1, 2, and 14 days with exposure to lower concentrations at the longer time periods. All three concentrations of the three insecticides produced liver damage. In fish exposed to 0.37 mg/l diazinon, vacuolation appeared within 1 day, while 8.0 mg/l dimethoate and 1.05 mg/l methyl parathion produced vacuolation only after 4 and 14 days, respectively.

The histopathological effects of malathion on the liver of the murrel (*Channa punctatus*) were investigated by Dubal and Shah.[149] Fish were exposed to concentrations ranging from 1 to 4 mg/l (96-hour LC_{50}, 2.5 mg/l) for 5–40 days. Malathion produced hepatopathy in all exposed animals. Hepatic lesions were characterized by the destruction of cyloplasmic and nuclear material, nuclear disarray, and shrinkage leading to damage of the protoplasmic material and vacuolization of the cells. Damage was greater at higher concentrations and longer exposure periods. Along with necrosis of the cells there was coagulation of blood in sinusoids, and the connected blood capillaries were filled with coagulated blood.

Csepai[150] studied the histopathological effects of four pesticides on the kidney of young carp. The fish were exposed to three insecticides, Anthio 40 EC, Satox 20 WSC, and Basudin 10 G, at concentrations of 20, 8, and 50 mg/l, respectively, and the herbicide Gramozone at a concentration of 15 mg/l. Basudin did not cause any lesions. For the other pesticides, histological hydropic and vascular degeneration and pycnosis were found. Gramozone caused intensive ring-like straining of the tubular membrane in the Bowman's capsule.

The effects of fenthion (50EC) on the gills of two cichlid fishes (*Herotilapia multispinosa* and *Tilapia leucosticta*) were investigated by Jauch.[151] Fenthion caused hyperplasia and separation of the respiratory epithelium in the gills of *Herotilapia multispinosa*. After a 96-hour exposure to 2.8 mg/l, 19% of the fish died and the surviving fish had severe lesions. After the surviving fish were returned to clean water for 28 days, the intoxicated gills barely differed from control gills. *Tilapia leucosticta* were more resistant to the insecticide.

The teratogenic effects of carbaryl, marathion, and parathion on developing medaka embryos exposed in static tests were investigated by Solomon[152] and Solomon and Weis.[153] The primary site of action of these insecticides was the circulatory system. Significant effects on circulatory anomalies were produced at concentrations of $\geq$5 mg/l carbaryl, $\geq$20 mg/l malathion, and $\geq$3 mg/l parathion. The overall percentage of circulatory abnormalities and their severity increased with increasing time of exposure. At combinations of low concentrations there was a slightly greater-than-additive effect. At higher concentrations both additive and antagonistic effects on circulatory abnormalities were found.

Grove and Moller[154] demonstrated the potential for using electric fish as a tool to monitor pesticides in water. The temporal pattern of electric organ discharges emitted by the mormyrid fish, *Brienomyrus niger*, was affected by both dieldrin and malathion at concentrations of 1 μg/l.

Herbicides. Gretch *et al.*[155] identified the herbicide 2,4,6-trichlorophenyl-p-nitrophenyl ether in rainbow trout (*Salmo gairdneri*) imported from Japan. The original trout sample contained 2.2 μg/g in the edible portion. Two subsequent samples contained residual levels of 0.6 and 0.2 μg/g. The structural elucidation, GLC characteristics, and recovery data for this herbicide were given. The effects of simazine treatment on channel catfish (*Ictalurus punctatus*) and bluegill (*Lepomis macrochirus*) production in ponds were investigated by Tucker and Boyd.[156] Application of 13.4 kg/ha of simazine to the bottom of channel catfish ponds 1

day before filling and stocking resulted in an extended period of low DO, a significant reduction in channel catfish yield, and a poorer feed conversion by the fish fed daily at 3% of body weight. A single application of 1.5 mg/l of simazine to the water of fertilized bluegill ponds also decreased DO, but the bluegill yield was not significantly different from the yield in the control ponds.

Spacie and Hamelink [157] described the behavior of trifluralin (TRF) in a natural fish population and determined the rate of TRF uptake and depuration in a laboratory study using fathead minnows (*Pimephales promelas*). TRF concentrations in the fat of 11 species collected at various stations were measured, with the high concentration being 440 μg/g of fat. In general, concentrations were greater in fish collected from the upstream stations below the industrial outfall. Major differences in residue levels with species, body size, or feeding habits were not apparent. The BCF for three species of fish based on an estimated mean river water concentration of 1.8 μg/l of TRF varied from 1 800 for the golden redhorse (*Moxostoma erythrurum*) to 6 000 for a minnow species.

The acute effect of the sodium salt of 2,4-D on embryos and larvae of bleak (*Alburnus alburnus*) exposed to 25–3 200 mg/l for 12–48 hours was investigated by Biro.[158] The high concentration of 2,4-D delayed or stopped the development of embryos in their early stages, caused behavioral changes, and morphological alterations. The 48-hour LC_{50} for the embryos and larvae were 12.9 mg/l and 51.6 mg/l, respectively. The acute toxicity of 11 herbicides to the mosquitofish (*Gambusia affinis*) in static tests was determined by Johnson.[159] The 96-hour LC_{50} values ranged from 0.35 mg/l for fenoprop to 19 100 mg/l for 2, 2-DPA.

Rao and Dad [160] reported the acute toxicity of some herbicides to fish and ectoprocta. The 96-hour LC_{50} values of seven static bioassays using three herbicides and five species of fish ranged from 3.1 mg/l of tagazine for *Sarotherodon mossambica* to 104.4 mg/l of aresin for *Channa punctatus*.

Studies were made to determine the acute toxicity of glyphosate, the isopropylamine salt of glyphosate, the formulated herbicide Roundup, and the Roundup surfactant of four aquatic invertebrates and four fish species.[161] The 96-hour LC_{50} values for Roundup varied from 2.3 mg/l for the fathead minnow to 13 mg/l for the channel catfish. Rainbow trout were intermediate in sensitivity. Technical-grade glyphosate, the active ingredient of Roundup, was less toxic than Roundup or the surfactant. Roundup was more toxic to rainbow trout and bluegills at higher test temperatures, and more toxic at pH 7.5 than at pH 6.5. Eyed eggs of rainbow trout were the most resistant life stage, and sensitivity increased as the fish entered the sac-fry and swim-up stage. No changes in fecundity or gonadsomatic index were found in adult rainbow trout treated with 2 mg/l of isopropylamine salt or Roundup. Rainbow trout did not avoid 10 mg/l of the isopropylamine salt.

Miller *et al.*[162] investigated the effects of static exposure of coho salmon (*Oncorhynchus kisutch*) and guppies (*Poecilia reticulata*) to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in water. The no-effect level on food consumption of coho salmon after 24, 48, and 96 hours of exposure and 40 days of postexposure was between 5.4 and 0.54 ng/g fish (5.6 and 0.56 ng/l). After exposure of coho salmon for 24, 48, and 96 hours and 114 days postexposure, the no-effect level on survival and growth was also between 5.4 and 0.54 ng/g fish. The critical duration of exposure time for growth of coho salmon was between 6 and 12 hours at an exposure level of 5.4 ng/g fish.

Woodward [163] assessed the hazard of picloram to cutthroat (*Salmo clarki*). The test was initiated using fish 3 days after hatching and continued for 60 days. The maximum exposure concentration not affecting survival and growth ranged from 290 μg/l for the first exposure period to 48 μg/l for the fourth exposure period. At the exposure regimen of 790 μg/l for the first exposure period and 76 μg/l for the fourth exposure period, survival of alevins and fry was similar to survival of control fish, but the final weight of the fry was significantly less than that of the controls.

Another study investigated the toxicity of butyl, isooctyl, and propylene glycol butyl esters of 2,4-D to cutthroat trout and lake trout (*Salvelinus namaycush*).[164] The 96-hour LC_{50} values and 61-day embryo/larval MATC values are listed in Table I. Both 2,4-D BE and 2,4-D PGBEE were more acutely toxic at lower temperatures, but pH and hardness did not influence toxicity. Both 2,4-D BE and 2,4-D PGBEE at a concentration of 500 μg/l caused 100% mortality to cutthroat alevins under a simulated field application,

while a concentration of 100 μg/l did not influence survival or growth.

Sublethal effects of 2,4-D and 2,4,5-T (butoxyethanol esters) on bluegill, *Daphnia pulex*, and *Chironomus* species were examined by Sigmon.[165] Exposure to 3 mg/l of either compound to small fish for 60 minutes or large fish for 30 minutes did not influence O_2 consumption of bluegills in a closed system at 20°, 25°, and 30°C. Residue levels of 2,4-D in whole fish were below the detection limit of 0.05 μg/g. Residue levels of 2,4,5-T were highest (0.12 μg/g) at 25°C and lowest (0.06 μg/g) at 30°C.

Some physiological and biochemical effects of the herbicide yalan (S-ethyl-N-hexamethyleneminothyocarbamate) on carp (*Cyprinus carpio*) were reported by Vrochinskij and Zemkov.[166] Yalan decreased the intoxication locomotor activity and glucose content of the blood and increased ammonia, urea, and lipid hydroperoxides in the white dorsal muscle.

Ochiai and Kubota [167] investigated the mode of action of molinate in carp. The toxic effect was caused by hemorrhagic anemia resulting from hemorrhages occurring mainly in the gills. Except for increased numbers of immature erythrocytes, no cytological change was observed in the blood of anemic carp. Bleeding time, plasma prothrombin time, and partial thromboplastin time were increased with increased exposure time. Both Vitamin K3 and Vitamin K4 were effective in preventing hemorrhagic anemia, while exposure to 0.8 mg/l of nifulpyrinal increased hemorrhagic anemia of fish exposed to molinate.

Another study to determine the effects of benthiocarb on carp showed that carp surviving lethal concentrations of 1.8 and 1.0 mg/l were anemic.[168] The anemia was expressed as a decrease in RBC count, hemoglobin volume, and hematocrit value. In both anemic and nonanemic carp that had been exposed to benthiocarb, spherical bodies were observed in the liver. The toxic effects of this herbicide on carp were anemia, degeneration of hepatic cells, and hemorrhage in the intestine.

Piscicides and anesthetics. The influence of temperature, pH, turbidity, ultraviolet light, and aquatic vegetation on the acute toxicity of 2-(digeranylamin)-ethanol, a prospective piscicide, to the fathead minnow (*Pimephales promelas*), rainbow trout (*Salmo gairdneri*), and brown trout (*Salmo trutta*) was evaluated by Launer and Bills.[169] Toxicity was reduced in cold water, in acid water, in turbid water where the turbidity was produced by bentonite or Crowley silt loam, and in water containing water weed (*Elodia* sp.). Toxicity was not changed by exposure of the chemical to ultraviolet light, by the presence of duckweed (*Lemna* sp.), or by turbidity-produced kaolin or barium sulfate. The toxicity of the compound was reduced by oxidation with potassium permanganate and calcium hypochlorite or by reduction with sodium thiosulfate.

Dwyer *et al.*[170] compared the chronic effects of 3-trifluoromethyl-4-nitrophenol (TFM) on brook trout (*Salvelinus fontinalis*) under conditions of continuous exposure and under conditions simulating the use of TFM for the control of the sea lamprey (*Petromyzon marinus*). In the continuous exposure study, survival and growth of the second-generation fish was the most sensitive effect. The survival and growth of these fry exposed to 1.6 mg/l for 90 days was significantly lower than the survival and growth of control fry. Residue analysis of TFM in the fillets from fish exposed for 137 days ranged from 0.01 μg/l for fish exposed to 0.4 mg/l to 0.11 μg/g for fish exposed to 16 mg/l. Under use-pattern conditions of exposure, adult fish exposed for 12 hours to 18 mg/l suffered significantly greater mortality than control fish.

The uptake, distribution, and elimination of [^{14}C]TFM by various tissues of largemouth bass (*Micropterus salmoides*) was measured by Schultz *et al.*[171] Maximum concentrations of radioactivity occurred in 8 hours in brain and muscle and in 12 hours in blood, liver, kidney, and head plus viscera of fish exposed to 1 mg TFM/l. In fish exposed to 1 mg/l for 12 hours and returned to clean water, the concentration of radioactive materials decreased with time throughout the 72-hour elimination study. TFM was not detected in muscle after 12 hours.

Srivastava and Srivastava [172] investigated the fungicidal activity of malachite green oxalate for fish disease control. The results of *in vitro* studies showed that the lowest effective fungicidal concentration was 0.5 mg/l for four fish-pathogenic isolates of saprolegniaceous fungi. This concentration was toxic to three species of fishes (*Puntius sophore*, *Colisa lalia*, and *Anabas testudineaus*). Tests conducted with *Catla catla*, a major food fish of India, showed that toxicity varied with the weight of the fish, with small fish being more sensitive.

Sills and Allen [173] conducted a study to determine whether residues of formaldehyde occurred in channel catfish (*Ictalurus punctatus*), largemouth bass, coho salmon (*Oncorhynchus kisutch*), and rainbow trout exposed to formalin at use-pattern concentrations for the control of external parasites. After 24 hours, <5 μg/g of formaldehyde was detected in muscle, liver, or blood plasma in fish exposed to 111 mg/l of formaldehyde. In a simulated pond study, channel catfish and largemouth bass exposed to 13 mg/l formaldehyde for 24 and 72 hours showed no detectable residues.

Abnormalities in the body curvature of the carp (*Cyprinus carpio*) after intramuscular injection of paraldehyde, tertiary butyl alcohol, and butanol were observed by Jain and Durve.[174] There was a long-lasting curing of the body posterior to the dorsal fin after injection of tertiary butal alcohol or butanol. The abnormalities affected locomotion and speed of swimming.

A test to determine whether Furanace-10, a broad-spectrum chemotherapeutic agent, added to anesthetic solutions of MS 222, quinaldine, and a mixture of these two compounds would influence survival of chinook salmon (*Oncorhynchus tshawytscha*) was conducted by Matthews.[175] Fish exposed for 1 hour to 10 mg/l of Furanace-10 added to each of the three anesthetic solutions survived as well as control fish not exposed to the head Furanace-10.

Post [176] discussed the advantages and limitations of the use of carbonic acid as an anesthetic agent for fish and other aquatic animals. Carbonic acid was found to be an inexpensive and easily obtainable anesthetic. The most satisfactory level of carbonic acid for anesthetizing fish was between 150 and 600 mg/l. Ferreira *et al.*[177] compared the anesthetic potency of benzocaine hydrochloride and MS 222 at concentrations of 50, 80, and 100 mg/l to carp and Mozambique mouthbrooder (*Sarotherodon mossambica*). The anesthetic induction times for benzocaine hydrochloride were generally shorter than for corresponding concentrations of MS 222. Recovery time was prolonged with increasing concentrations of both compounds. The concentration of free ethyl p-aminobenzoate and free ethyl m-aminobenzoate in the blood of both species showed an increase with increasing concentrations of both anesthetic agents. The effect of MS 222 and R 7464 (Propoxate) on O_2 consumption of the tench (*Tinca tinca*) was measured by Jirásek *et al.*[178] Oxygen consumption of the fish at 10° and 20°C exposed to 5, 20, and 50 mg/l of MS 222 was reduced by 26–54%. Oxygen consumption of the fish at 10° to 20°C exposed to 0.25, 0.33, and 0.50 mg/l of R 7464 was reduced 13–74%. Both compounds reduced the excretion of metabolic products as measured by a BOD test.

Inorganics. Arsenic concentrations in fish from Chautauque Lake were investigated by Foley *et al.*[179] Lake water samples ranged from 22.4 to 114.8 μg As/l. Fish concentrated but did not magnify As. The black crappie (*Pomoxis nigromaculatus*) had the highest levels with a mean of 0.7 μg As/l, while perch (*Perca flavescens*), muskellunge (*Esox masquinongy*), and largemouth bass (*Micropterus salmoides*) had the least, 0.02–0.13 μg As/l. Kent and Johnson [180] analyzed fish, water, and sediment for Hg, As, and Cd in American Falls Reservoir, Idaho. Levels in water ranged from 1.5 to 3.3 μg As/l, 0.25 to 1.78 μg Hg/l, and 1.0 to 24.5 μg Cd/l. Five species of fish showed mean values ranging from 0.17 to 0.37 μg Hg/g and 0.15 to 0.51 μg Cd/g. No As was found in the fish. Wiener and Giesy [181] examined the trace metal concentration and uptake of fish from a highly organic soft-water pond (Skinface Pond) in the southeastern coastal area. In general, fish were found to contain levels of Cu, Mn, Pb, and Zn similar to other reported values. When fish were put in the lake from an outside source they first accumulated some metals and then returned to near background levels. Murphy *et al.*[182] performed Zn and Cd analysis on 11 species of fish from an industrially contaminated lake. Most species accumulated the metals significantly higher than background. Higher trophic levels had generally lower levels of these trace elements.

Hattula *et al.*[183] surveyed several species of fish for Hg from various areas of a large freshwater lake in Finland. Twelve species all utilized as food by humans were included. The mean concentration found was 0.65 μg Hg/g wet weight of muscle, with 98.7% being in the methylated form. An accumulation coefficient between Hg water content and carp (*Cyprinus carpio* L.) muscle of 913 ± 161.7 was found by Chodyniecki and Protasowicki [184] from a survey of 52 fish from three different waters. The male guppy (*Lebistes reticulatus*) was used to study the transfer of Hg from the sediments to fish.[185] Uptake by

the fish that could accumulate Hg only from water was 0.02 μg Hg/g, compared with 0.18 μg Hg/g from those that had access to sediments and water. Values were determined by counting live fish using ^{203}Hg tracer.

An analysis of fish for Hg from 208 lakes in northern Saskatchewan was reported by Murray.[186] Only 25 of the examined lakes had fish with 0.5 μg Hg/g or greater. In general, those fish at the top of the food chain, that is, walleye (*Stizostedion vitreum*) and northern pike (*Esox lucius*), contained the most Hg. Penaz *et al.*[187] analyzed muscle tissue from a variety of fish from the Jihlava River in Czechoslovakia. Mercury levels varied from 0.07 to 0.81 μg Hg/g. Older fish usually had higher levels. Fish exposed to ^{238}Pu from a spill were analyzed by Kennedy and Bartelt.[188] The piscivorous fishes found had less Pu than the bottom feeders, with black bullhead having the highest levels. Within 24 hours uptake of Pu was found to occur at the same level as found after 6 months. Muscle tissue was found to have relatively the least, with the concentration order being GI tract > gill > liver > bone > skin > muscle.

Singh and Ferns[189] fed 30% dried sludge from an activated wastewater treatment plant as part of the diet to juvenile rainbow trout (*Salmo gairdneri*). Some elevation of levels of Cr, Fe, Ni, and Pb was noted, but the mean values for all groups fell within the range for the controls. Oglesby *et al.*[190] concluded that negative redox potentials allowing uptake of Mn and humic acids by rainbow trout, brown trout (*Salmo trutta*), and brook trout (*Salvelinus fontinalis*) caused a massive fish kill below a strongly stratified reservoir.

The levels of Cu and Zn from an acid mine waste that were lethal to eggs and fry of steelhead trout (*Salmo gairdneri*) were determined by Finlayson and Ashuckian.[191] The LC_{50} values were determined in this complex waste with two ratios of Cu to Zn. In 1:4 the overall 60-day LC_{50} was at the level of 0.20 mg Zn/l and 0.043 mg Cu/l. In 1:12 exposure, the levels were 0.56 mg Zn/l and 0.035 mg Cu/l. Incipient lethal levels of 0.03 mg Zn/l and 0.01 mg Cu/l were also reported.

High levels of Cu (93–809 μg/g, wet weight) were found in the livers of brown trout below a copper-containing wastewater outfall.[192] The author speculated that the Cu came from snails that also contained high Cu values, since snails were eaten as the main food source by the trout. Murphy *et al.*[193] analyzed field samples of bluegill (*Lepomis macrochirus*) and largemouth bass from two areas contaminated with Cd and Zn. Mean dissolved metal levels of 17.3 μg Cd/l and 293 μg Zn/l in the high areas and 0.9 μg Cd/l and 52.4 μg Zn/l in the low areas were found. Residues (μg/g) in bluegills were (high) 3.4 Cd and 220 Zn and (low) 0.01 Cd and 140 Zn. Bluegill sunfish concentrated Cd approximately six times more in fillet samples than did largemouth bass. The mean difference in Zn concentration was less than 2.

Radford and Graveland[194] showed that coal mine effluents had only a limited effect on receiving streams and metal accumulation in fish in southern Alberta. Precipitation of iron oxides was the most damaging effect. Concentrations of Cu, Pb, and Zn did not exceed levels considered safe for consumption in any fish analyzed. Scullion and Edwards[195] investigated the movements of hatchery-reared rainbow trout and brown trout following a release into a small coal-waste polluted river in South Wales. Electrofishing and angler catch-return data indicated extensive downstream movement of both species. After 2 weeks about 50% had either been caught or had moved out of the river; after 2 months, only 10% remained.

The effect of coal ash on the biota of a swamp drainage system was reported by Cherry *et al.*[196] Siltation, decreased pH, and metal concentrations were most effective in changing the biota. *Gambusia affinis* was the only fish present and concentrated Cu, Zn, and Se. Elser and Schreiber[197] studied the fish populations in Rosebud Creek, a prairie stream flowing through the Fort Union Coal Basin in southeastern Montana, to determine whether there were any immediate effects and to provide baseline data for assessing possible future effects from accelerated coal mining or coal combustion in the area. No apparent effects of the coal related activities were noted. The species composition and fish distribution were representative of the other streams in the area.

Acute toxicity and embryo-larval tests with rainbow trout, fathead minnows (*Pimephales promelas*), and *Daphnia pulex* by DeGraeve and Bergman[198] demonstrated that waters condensed from underground coal gasification (UCG) and from underground retorting of oil shale were toxic when diluted down to 0.57–0.01%. The principal constituents influencing toxicity were believed to be inorganics in the case of oil retort water and ammonia and phenolics from UCG condenser water.

Birge [199] performed embryo-larval bioassays with goldfish, trout, and toads to establish a comparative toxicological ranking for 22 elements in coal and fly ash. Silver, Cd, Cr, Hg, Ni, and Pb were among the 12 most toxic for all three species, and Al, As, Co, Cu, Ge, V, and Zn were included in the most toxic group for trout and goldfish (LC_{50}s were 0.1 mg/l or less). Spotila and Paladino [200] found that newly hatched muskellunge were quite insensitive to As (>5 mg As/l). Toxicity was found to be the greatest at the swim-up stage and then decreased with increased age of the fish.

Continuous-flow toxicity tests were conducted to determine the relative tolerances of newly hatched alevins, swim-up alevins, parr, and smolts of chinook salmon (*Oncorhynchus tshawytscha*) and steelhead to Cd, Cu, and Zn.[201] Newly hatched alevins were much more tolerant to Cd and, to a lesser extent, to Zn than were later juvenile forms. However, the later progression from swim-up alevin through parr to smolt was accompanied by a slight increase in metal tolerance.

Studies by Beattie and Pascoe [202] gave a time and concentration positive correlation with Cd versus rainbow trout eggs. Values of up to a 50-fold increase in toxicity at 100-hours of exposure at 10 mg/l were noted. Kariya *et al.*[203] reported a 48-hour LC_{50} of 5–10 Cd/l to goldfish (*Crassius auratus*). Combinations of Cd and CN in plating wastes resulted in 48-hour LC_{50} values from 0.3 to 3.5 mg Cd/l and 0.2 to 0.7 mg CN/l. Concentrations of Cd were found in the kidney and hepatopancreas after death at levels of 1–3.5 μg/g of tissue.

Servizi and Martens [204] produced a report on the effects of Cu, Cd, and Hg on the eggs, alevins, and young fry of sockeye (*Oncorhynchus nerka*) and pink salmon (*O. gorbuscha*). Mortality, hatching, and growth of sockeye salmon during the egg-to-fry stage were not affected by continuous exposure to 5.7 μg Cd/l, but 8 μg Cd/l was lethal to 50% of the fish in 168 hours. The incipient lethal level for Cu was between 37 and 78 μg/l for sockeye salmon and between 25 and 55 μg/l for pink salmon during the egg-to-fry stage. Copper inhibited egg capsule softening and was concentrated by eggs, alevins, and fry in proportion to exposure concentrations. Continuous exposure of eggs to 2.5 μg Hg/l caused malformed embryos at hatching, which was the most sensitive effect observed. Mercury was concentrated by sockeye and pink salmon in proportion to exposure concentration during the egg-to-fry stage.

Chakoumakos *et al.*[205] ran a series of 96-hour bioassays with Cu on cutthroat trout (*Salmo clarki*) under different conditions of hardness, pH, and alkalinity under constant flow conditions. The LC_{50} values ranged from 0.4 mg/l total Cu at high hardness and alkalinity and pH to 0.02 mg/l at low values for the test parameters.

Copper toxicity to young life stages of sockeye salmon was studied by Davis and Shand.[206] The 96-hour LC_{50} values are reported in Table I. Copper-exposed fish were found to be more sensitive to a seawater challenge than unexposed fish. Ninety-six-hour renewable static bioassays were conducted by Lewis [207] on longfin dace (*Agosia chrysogaster*) with Cu, Zn, Mn (Table I), and their combinations in hard water. The LC_{50} values for the combinations were 0.21 Cu/l plus 0.28 Zn/l, and 0.45 mg Cu/l plus 64 mg Mn/l. The toxicity of Cu to bluegill and channel catfish (*Ictalurus punctatus*) in hard water (260 mg $CaCO_3$/l) was determined by Richey and Roseboom.[208] The 14-day LC_{50} for soluble Cu was 2.5 mg Cu/l for bluegill and 1.2 mg/l for catfish. Chapman and Stevens [209] determined the acute LC_{50} values for Zn, Cd, and Cu to adult male steelhead trout and coho salmon (*Oncorhynchus kisutch*, Table I). Extended Cd exposure times (215-hour LC_{50} of 3.7 μg Cd/l for coho and 408-hour LC_{50} of 5.2 μg Cd/l for steelhead trout) were reported, as Cd appeared to have a latent effect, with little toxicity at 96 hours.

Other studies determined the acute toxicity of Zn to rainbow trout and brook trout [210] and *Rasbora daniconius*.[211] The 96-hour values are shown in Table I. Heisinger *et al.*[212] experimented with the effect of Se on inorganic Hg toxicity to the goldfish. The 94-hour LC_{50} was reduced from 0.35 mg Hg/l to between 0.44 and 0.5 mg Hg/l with equimolar, simultaneous, or pretreatment Se application. Paradoxically, the uptake of Hg was enhanced by Se under the same conditions, from 27.83 to 46.03 μg Hg/g dry weight. The acute toxicity of Hg to *Channa punctatus* was determined by Sastry *et al.*[213] The 96-hour LC_{50} for $HgCl_2$ was 1.8 mg/l.

Carp eggs and fry were exposed to Ni by Blaylock and Frank.[214] The LC_{50} values for egg-exposed larvae, 72-hour posthatch larvae, and 96-hour posthatch larvae were 6.10, 8.46, and 6.16 mg Ni/l, respectively. Srivastava and Mishra [215] determined the acute toxicity

of Pb to *Colia fasciatus*. The 96-hour LC_{50} value was 19 mg/l. Ferreira and Wolke [216] determined the acute toxicity of Pt to coho salmon. The 24-, 48-, and 96-hour values are shown in Table I. Adult flagfish (*Jordanella floridae*) exposure to vanadium (V) resulted in a 96-hour LC_{50} of 11.2 mg V/l as reported by Holdway and Sprague.[217] Larval exposures resulted in 28-day LC_{50} values of 1.13–1.88 mg V/l, with larger animals being more resistant. A concentration of 0.04 mg V/l produced no deleterious effects.

Guppies and goldfish were exposed to various V compounds in deionized water in another study.[218] The 96-hour LC_{50} was between 3 and 20 mg V/l for goldfish and 0.5 and 5 mg V/l for guppies. Vanadyl sulfate was the most toxic form tested. Extension of the time to 144 hours increased the toxicity of most of the compounds about three to five times. Yearling carp were exposed to sublethal levels of sodium fluoride (25 mg NaFl/l) by Prochnow.[219] A 63-day exposure showed no visible changes in the vertebrae column; however, fluoride was concentrated about 10 times over control values during this period.

Smith and Heath [220] tested rainbow trout, golden shiner (*Notemigonus crysoleucus*), bluegill, goldfish, and channel catfish against KCN, $CuSO_4$, $ZnSO_4$, and $K_2Cr_7O_4$ at 5°–30°C for all except the trout, which were tested at 5°–18°C. It was concluded that, except for the goldfish, the test temperature had little effect (less than a factor of 3). Goldfish toxicity changed as much as 6-fold over the same range of temperatures.

The effects of chronic exposure of sockeye salmon to Zn during their freshwater residency was studied by Chapman.[221] A concentration of 242 μg Zn/l produced no adverse effects on survival, fecundity, growth, or survival of subsequent smolts in seawater. An application factor of 70.15 was reported between the 96-hour LC_{50} and the "safe" concentration in soft (35 mg $CaCO_3$/l) water.

A determination of toxicity, food intake, growth, and bioaccumulation of Zn on juvenile Atlantic salmon (*Salmo salar*) was made by Farmer *et al.*[222] The 21-day LC_{50} values ranged from 1.6 to 0.35 mg Zn/l. Smaller (5 g) fish were less sensitive than larger (16–17 g) fish. Food intake was initially lowered inversely to sublethal concentration but returned to control levels after 30 days of an 80-day experiment. Concentration of 0.56 mg Zn/l and lower did not affect growth. Fish fed to satiation daily accumulated less Zn than those on a set ration, apparently because the muscle and fat took up less Zn and were a larger percentage of well-fed fish.

Benoit and Holcombe [223] conducted a life-cycle exposure with Zn on the fathead minnow. Adverse effects were found with the larvae at all levels above 295 μg Zn/l. A concentration of 145 μg Zn/l caused egg adhesiveness and egg strength changes that were significantly different from the controls. A β-generation study to determine the effect of Zn on brook trout resulted in a MATC between 534 and 1 360 μg Zn/l.[224] Several tissues, gill, kidney, liver, and bone accumulated Zn, but muscle did not. No generation differences were found. Rainbow trout juveniles and subadults were found not to accumulate Cu at the levels and conditions normally used for algae treatment in hard water lakes.[225]

Sorensen *et al.*[226] exposed green sunfish (*Lepomis cyanellus*) to 60 mg As/l from sodium arsenate for 2, 4, and 6 days. Arsenic uptake was highest in the gall bladder and bile, followed by the liver, spleen, and kidney. Values ranged from 159, 49, 23, and 14 μg/g, respectively, after a 6-day exposure.

Cadmium uptake studies with *Gambusia affinis* and *Lepomis macrochirus* and food were conducted by Giesy *et al.*[227] The authors found most of the uptake was directly from the water, with uptake attributed to food being nonsignificant in all cases. Roberts *et al.*[228] exposed brown trout and rainbow trout to Cd at 27 and 9 μg/l, respectively. The gill, kidney, and liver had values of 3.0–0.3 μg/g after 2 weeks and from 17.7 down to 5.2 μg/g after 3 months. Rainbow trout accumulated more Cd in most tissues. Sangalang and Freeman [229] reported that fish exposed to 1 μg Cd/l for 93 days accumulated Cd in the gills and posterior kidneys to levels of 0.9 μg Cd/g wet weight. No other tissue exceeded 0.25 μg Cd/g at the end of the exposure period.

Nabrzyski and Gajewska [230] reported an analysis of freshwater and saltwater fish for Zn and Cd. It was found that the Zn/Cd ratio was always 170 parts Zn to 1 part Cd. Freshwater means were 904 μg Zn and 5.1 μg Cd per 100 g tissue, and saltwater means were 817 μg Zn and 48 μg Cd per 100 g tissue. A high level of 26.4 μg Cd per 100 g was found, but the ratio remained the same.

Uptake of methyl mercury and inorganic Hg from food by the rainbow trout was reported in a study by Jacobs.[231] The authors found

that 7 μg/g of methyl mercury in the feed resulted in a muscle value of 1.04 μg/g in 8 days. Levels of 1.02 μg/g food resulted in 0.67 μg/g in muscle, 1.06 μg/g in liver, and 1.38 μg/g in the kidney, while inorganic Hg exposure at similar levels had no significant uptake. Svobodova [232] exposed carp to organic Hg via the food at 17.5 μg/g of food. Accumulations ranging from 2.65 μg/g in kidney, 0.81 μg/g in hepatopancreas, and 0.12 μg/g in muscle were found after a 6-month exposure. After 18 months depuration values declined, but the total amount of Hg in the fish was unchanged during this period.

Cember *et al.*[233] compared the effect of methyl mercury concentration in water and the effect of temperature differences on bioconcentration in bluegills. BCF was found to increase exponentially with temperature at the rate of 0.066 per °C. The BCF was not affected by water concentration of methyl mercury. Phillips and Buhler [234] exposed rainbow trout to combinations of food, water, and food and water contaminated by methyl mercury. When compared to unexposed controls, food and water exposures were equally effective in producing accumulations of Hg in the test fish. The uptake was linear with time, with a 0.97 coefficient of determination for all treatments.

An investigation of Pb uptake in the coho salmon as influenced by Ca was undertaken by Varanasi and Gmur.[235] Lead concentrations of 0.13 and 0.21 mg/l were used, and 8.4 mg Ca/l in the diet significantly reduced Pb uptake by the water and food route. Increased Ca (6.2 mg/l) in the water reduced the uptake rate in gill, blood, skin, and skeleton, but not in the liver and kidneys, when compared with a concentration of 2.6 mg Ca/l in the water.

Paladino and Spotila [236] reported that As levels between 5 and 0.05 mg/l depressed the critical thermal maxima (CTM) for muskellunge slightly (about 1°C for 5 mg/l exposure), but exposed animals did not recover from the CTM depression concomitant with swim-up as did the controls. Also, 0.05 mg/l killed 50% of the fry after a 5-day exposure.

The effects of several heavy metals on smolting and survival during seawater reentry were reported.[237] Copper exposure and Hg caused mortality upon seawater reentry of freshwater exposed smolts. Combinations of Cd with Cu and Zn with Cu produced synergistic effects. Cadmium, Cr, Ni, and Zn alone caused no additional mortality upon seawater reentry.

Passino and Cotant [238] exposed allantoinase, an enzyme of lake trout (*Salvelinus namycush*), to several heavy metals and DDT and PCB (Aroclor 1254). During a 10-minute *in vitro* exposure, allantoinase was not inhibited by DDT or DDE up to 10 μg/g or PCB up to 7 μg/g. Fifty percent reduction of enzyme activity was noted at 6 mg Cu/l, 34 mg Hg/l, and 52 mg Pb/l.

Shaffi [239] exposed three freshwater teleosts, *Labea robita*, *Ophicephalus punctatus*, and *Clarius batrachus*, to $CuSO_4$. All species showed a similar reaction. A 3-hour exposure to 5, 10, and 15 mg $CuSO_4$/l produced reduction of the glycogen content in liver, muscle, and brain and increased glycogen in the kidney. Changes were concentration dependent. Waiwood and Beamish [240] conducted growth studies with rainbow trout exposed to varying Cu, hardness, and pH. Fish were fed a set ration and exercised. All combinations down to 4 μg Cu/l depressed the growth rate initially by up to 20% for 10 days. All fish regained the normal control growth rate except where pH was maintained below 6.0. Rainbow trout were exposed to a sublethal (10 μg Cu/l) dose of Cu and then challenged with IHN virus.[241] The results showed that Cu was more toxic when combined with this virus.

Dubale and Shah [242] found that 0.01 mg $Cd(NO_3)_2$ per litre caused necrosis of hepatic cells in *Channa punctatus* after a 24-hour exposure. Within approximately 17 days, some recovery was seen in the lower concentrations (0.03–0.01 mg/l), but this was reversed after Day 40 with increasingly severe results. Koyama *et al.*[243] administered Cd in the food to carp and porgy (*Pagus major*). Changes in kidney structure were most pronounced in the epithelial cells of the proximal segment of the renal tubules of both species. Changes included increase and enlargement of lysosomes and apical vacuoles, increase in coated vesicles and apical tubules, and dilation of smooth-surfaced endoplasmic reticulum.

Histological preparations of organs from *Heteropneustes fossilis*, a teleost fish, exposed to 6.8 mg/l of $CdCl_2$ for 30 days were examined by Sastry and Gupta.[244] Nearly all of the damage was found in the liver connective tissue, including liver cord disarray, enlargement and vacuolation of hepatocytes, accumulation of lipid, and focal necrosis.

Rainbow trout gained resistance to Cd toxicity when pretreated with lower levels.[245] The authors reported an increase of the 48-hour LC_{50} from <0.1 mg Cd/l for dilution water pretreatment to 0.11 and 0.15 mg Cd/l for alevins pretreated at 0.001 and 0.01 mg/l, respectively. Changes in tissue glycogen were reported by Shaffi [246] for *Labea robita, Ophicephalus punctatus,* and *Clarius batrachus* exposed to $Cd(NO_3)_2$ for 3 hours. In the liver and muscle, glycogen values fell at all exposures. Values (volumes of Cd) in the kidney and brain rose at 5×10^{-4} and 10×10^{-4}% and fell at 15×10^{-4}%.

Banerjee *et al.*[247] compared the effects of Cd on some of the enzymes of *Clarias batrachus,* an air-breathing fish, and *Tilapia mossambica,* a non-air-breather. Although the enzyme systems were changed in the same direction in both species, the amount of change varied between species. Uptake of Cd was much higher in the water-breathing fish.

A freshwater teleost, *Colisa fasciatus,* was exposed to Co by Srivastava and Agrawal.[248] The authors reported that 195 mg $CoCl_2$/l caused a decrease in small leucocytes and increased clotting time. Other blood factors were only slightly affected. Sugatt [249] observed that coho salmon exposed to Cr^{6+} in fresh water at a level of 0.5 mg/l for 2 weeks and then transferred to clean sea water had increased mortality and decreased resistance to *Vibrio anguillarum.* No mortality occurred in the fresh water.

The effect of an *in vivo* exposure of $HgCl_2$ on the activities of acid and alkaline phosphatases, glucose-6- phosphatase, lipase, and urease in the kidneys and ovaries of a teleost fish, *Channa punctatus,* was studied after 96 hours and after 30 days.[250] The activities of all the enzymes except acid phosphatase were significantly inhibited in both tissues at a LC_{50} concentration of 1.8 mg/l and a sublethal concentration of 0.30 mg/l. However, treatment for 96 hours resulted in more marked inhibition than treatment for 30 days. Acid phosphatase showed elevation in activity in the kidney after 96 hours. Another study found alterations in the activities of alkaline phosphatase, acid phosphatase, glucose-6-phosphatase amylase, trypsin, pepsin, amintripeptidase, glycylglycine dipeptadase, and carnosinase caused by exposure of *Channa punctatus* to a sublethal concentration of 0.30 mg $HgCl_2$/l for 20 days.[251] A fall in the activities of the three phosphatases was recorded except for alkaline phosphatase, which showed a slight elevation in activity in intestine and pryloric caeca. An increase in the activity of amylase and the two proteases was observed in all portions of the digestive system. The three peptidases revealed a decrease in activity. Exposure to $HgCl_2$ was found to disrupt the nitrogen metabolite balance of *Rutilus rutilus caspicus* in studies reported by Dokholyan and Akhmedova.[252] Some enzyme levels were lowered and others were raised. Dial [253] exposed Japanese medaka (*Oryzias latipes*) embryos to 80 μg CH_3HgCl_2/l. The resulting change in eye development correlated with other gross changes in the animal. Huckabee *et al.*[254] determined that methyl mercury was not formed *in vivo* in brook trout during exposure to inorganic Hg as nitrate. The authors also concluded that the methyl mercury normally found in wild fish comes from the food chain, as water levels are usually too low to cause much uptake in what is usually regarded as "clean" water.

Agrawal *et al.*[255] reported that 45 mg $NiSO_4$/l resulted in changes in six of the eight hematological parameters tested after 90 hours of exposure. The change was most marked in the reduction of small lymphocytes moving in the blood. The effect of $PbNO_3$ on liver, muscle, brain, kidney glycogen, serum glucose, and lactate was studied in nine species of fish by Shaffi.[256] Glycogenolysis was higher in major carp species than in catfish or murrels. The lowest level of glycogen was in muscle, followed by liver, kidney, and brain. There was an inverse relationship between the concentration of $PbNO_3$ and the fall in liver, muscle, and brain glycogen, whereas there was a direct relationship between Pb concentration and the levels of serum glucose and lactate. The effect of $PbNO_3$ on the activities of several enzymes in the kidneys and ovaries of a teleost fish, *Channa punctatus,* was examined by Sastry and Agrawal [257] after 96 hours and 30 days, respectively. All five enzymes in the two tissues were inhibited significantly at both the LC_{50} level and at a sublethal concentration. However, the inhibition produced after 30 days by the sublethal concentration was higher, indicating the cumulative action of Pb. Further, the inhibition of enzymes was more marked in the kidney than in the ovary.

Ozoh [258] found that exposure of eggs and larvae of *Brachydanio rerio* to a combination of Pb and Cu (36 μg/l) in distilled water caused various structural anomalies and also

reduced hatching success. Zinc exposure at sublethal levels reduced the brood size of the common guppy.[259] Concentrations of 0.36, 0.88, and 1.70 μg Zn/l reduced the brood size 24, 58, and 50% when compared to the control fish. Time between broods was not affected. Malformation in the young from Zn-exposed females was also noted. Judy and Davies[260] used the fathead minnow to develop a data set on the effect of Ca as $Ca(NO_3)_2$ on Zn toxicity. Calcium increased to 150 mg Ca/l reduced the toxicity of 6 mg Zn/l to 0 in a 96-hour static test.

Eggs and larvae of *Cichlasoma nigrofasciatum* were briefly exposed to low concentration of Zn and Cu by Ozoh and Jacobson.[261] The authors found that distilled water exposure to Cu ions had a severe effect on malformation, while Zn had no morphological effect but was more effective in preventing hatching. Changes in behavior of bluegill sunfish upon exposure to sublethal concentrations of Zn and Cd were reported by Henry and Atchison.[262] Increasing metal exposures were directly correlated with various behavioral changes, such as coughing, erratic swimming, and aggressiveness. In an effort to determine the cause of more rapid mortality from Zn under hypoxic conditions, Hughes and Flos[263] exposed rainbow trout to 10 mg $ZnSO_4$/l for 10 hours in hard water and static conditions. Results showed that there was no correlation between a hypoxic state and the final Zn concentration in the gill tissue. Singh and Singh[264] determined the effect of Zn and Cd on O_2 consumption in *Mystus vittatus*. The authors found that O_2 consumption was depressed 50% in 12 hours by 40 μg Zn/l and 3.4 μg Cd/l.

Organics. Norstrom *et al.*[265] analyzed organochlorine residues of coho salmon (*Oncorhynchus kisutch*), alewives (*Alosa pseudoharengus*), and smelt (*Osmerus mordax*) from pooled salmon stomach contents and herring gull (*Larus argentatus*) eggs, which they felt provided an integrated measure of organochlorine contamination of Lake Ontario. Coho salmon muscle residues were two to three times higher than residues in pooled alewife/smelt samples, while herring gull eggs had concentrations 40 to 60 times greater than coho levels. Coho muscle contained 5.8 μg PCB/g, 0.97 μg p,p′-DDE/g, 0.23 μg mirex/g, 0.11 μg photomirex/g, 0.11 μg p,p′-DDD/g, and other organochlorines between 0.015 and 0.097 μg/g.

Residue analyses of young-of-the-year spottail shiners (*Notropis hudsonius*) collected from sites on lakes Ontario, Erie, and St. Clair[266] showed this fish to be a useful biological integrator of organochlorine contamination in near-shore locations. Contaminant types and residue levels in fish generally reflected the regional land practices. All samples analyzed contained PCBs (59–844 ng/g) and DDT (32–244 ng/g, mostly as p,p′-DDE), and some also contained heptachlor epoxide, dieldrin, endrin, and chlordane.

Kent and Johnson[267] analyzed fish, water, and sediment samples from the American Falls Reservoir on the Snake River in southeastern Idaho for organochlorine contamination. Residues of TDE, DDE, and PCBs as high as 1.96, 2.79, and 28.74 μg/kg, respectively, were found in sediments; all water samples were below analytical detectability. Residues of TDE, DDE, and dieldrin in the flesh of sport fish were as high as 53.3, 67.2, and 160.4 μg/kg, respectively. The highest residue levels were found in a commercially fished sucker, *Catostomus ardens*, with 1.1 μg PCB/g, 781.7 μg TDE/kg, and 82.1 μg DDT/kg.

Composite fish samples from major U. S. watersheds showed that PCBs were in 93% of the samples at a concentration range of 0.3–140 μg PCB/g on a whole fish basis.[268] Of the samples, 53% were above 5 μg/g, the current FDA tolerance level; only 14% contained less than 2 μg PCB/g. Total DDT concentrations ranged from <0.05–4.53 μg/g. Hexachlorbenzene and chlordane components were identified in 19 and 36% of the samples, respectively. Numerous other organics were also identified but not quantitated.

Samples of burbot (*Lota lota*) liver and lake trout (*Salvelinus namaycush*) mesentery fat from the Churchill Falls Power Development area showed that PCB levels in the fish from a site at the outfall of the Churchill Falls Power Plant (the "tail race") were up to two orders of magnitude higher than those in fish from the general area above the power plant.[269] Above the plant, values ranged from 0.04 to 1.11 μg PCB/g wet weight with a mean of 0.51 μg PCB/g for burbot liver and 0.50 μg PCB/g for lake trout fat while values in burbot livers from the "tail race" ranged from 1.2 to 35.4 μg PCB/g with a mean of 17.1 μg PCB/g.

Wszolek *et al.*[270] netted lake trout ranging in age from 6 to 12 years from Cayuga Lake in central New York in 1978 and analyzed for PCBs and p,p′-DDE. Fresh weight concentrations ranged from about 4–5 μg PCB/g in 6-year-old fish to about 13 μg PCB/g in 12-year-

olds and from 1 to 2 μg p,p′-DDE/g in 6-year-olds and 3 to 4 μg p,p′-DDE/g in 12-year-olds. Compared to similar samples from Cayuga Lake in 1970, p,p′-DDE decreased considerably while PCB concentrations were about the same.

As part of a larger study of PCB contamination in the Hudson River, N. Y., Skea *et al.*[271] placed four species of fish, brown bullheads (*Ictalurus nebulosus*), yellow perch (*Perca flavescens*), pumpkinseeds (*Lepomis gibbosus*), and creek chubs (*Erimyzon oblongus*), in live cages in the Hudson River and analyzed the fish for Aroclor 1016. The mean PCB concentration in the Hudson River during the 14-day exposure period was 0.10–0.17 μg/l. The PCB residues were similar in the four species analyzed (Table I), and the concentrations were related to fat content and weight of the fish. Mean fish concentration was 2.6 μg PCB/g (range 1.8–3.8 μg/g), yielding a bioaccumulation factor between 15 000 and 26 000.

Zitko and Saunders[272] analyzed samples of Atlantic salmon (*Salmo salar*) eggs from four New Brunswick rivers for organochlorine residues and determined the hatchability of these eggs under normal hatchery conditions. Residues in the lipid of the eggs were 1.88–6.48 μg PCB/g, 0.086–0.159 μg hexachlorobenzene/g, 0.16–0.48 μg DDE/g, 0.34–0.41 μg p,p′-DDD/g, 0.20–0.46 μg DDT/g, 0.21 μg dieldrin/g, and 0.13–0.21 μg trans-nonachlor/g. Mean hatchabilities of the Atlantic salmon eggs (79.8–91.4%) did not correspond to the relative residue burden of the eggs. The authors concluded that the hydrocarbon levels were not high enough, singly or in combination, to affect hatchability.

PCB residues in five resident fish species from the Furans River in the south Jura, France, were analyzed by Keck and Raffenot.[273] Highest PCB levels occurred downstream from its confluence with the Arene River, known to be heavily polluted by domestic wastes. Two cyprinids, the barbel (*Barbus fluviatilis*) and the chub (*Leuciscus cephalus*), contained generally higher residues (1.3–2.7 μg PCB/g of muscle) than did the salmonids, brown trout (*Salmo trutta*), and grayling (*Thymallus thymallus*) (0.4–1.0 μg PCB/g of muscle). The accumulation of PCB varied with the fish tissues, and the liver always concentrated the most, followed by gills, eggs, and muscle. The authors found no correlation between the lipid content of the organ and its PCB content within a species.

Residues of resident and caged fish from the Pine River in Gratiot and Midland Counties, Mich., downstream from the Michigan Chemical Corporation Plant (the major PBB manufacturer in the U. S.), were analyzed as part of a study to evaluate the contamination of this river.[274] Significant levels of PBBs were detected in most fish species collected near the chemical plant ranging up to a maximum of 1.33 μg/g in carp (*Cyprinus carpio*) fillets. No PBBs were detected in fish from a reservoir upstream from the plant and above a barrier dam. Caged fathead minnows (*Pimephales promelas*) from two stations below the plant accumulated 1 μg PBB/g within 2 weeks. Water concentrations were below analytical sensitivity (0.1 μg/l) at one station and a mean of 3.9 μg/l (range: 0.9–9.8 μg/l) at the other, yielding bioconcentration factors of greater than 10 000 (Table I).

Fish obtained near dye or textile manufacturing locations in New York, New Jersey, Michigan, and Georgia were analyzed for selected aromatic amines by Diachenko.[275] Downstream of a dye plant in the Buffalo River, N. Y., carp fillets contained 10 ng 1-naphthylamine/g and 45–100 ng N-ethyl-N-benzylamine/g. Whole fish composite samples of minnows collected downstream in the Delaware River, N. J., contained 40 ng 1-naphthylamine/g.

Yurawecz[276] identified chlorinated trifluorotoluene (TFT) and chlorinated toluene residues in Niagara River, N. Y., fish. Gas-liquid chromatographic-mass spectrometric data of edible portions of fish revealed trace to 3.0 μg/g of 4-chloro-TFT, 2-chloro-TFT, 3,4-dichloro-TFT, 2,4-dichloro-TFT, 2,6-dichlorotoluene, 2,4,5-trichlorotoluene, as well as other TFT and toluene isomers. These toluenes had not been previously reported as residues in environmental samples.

Niimi[277] analyzed residues of hexachlorobenzene (HCB) in lake trout, rainbow trout (*Salmo gairdneri*), and coho salmon collected from Lake Ontario. HCB concentrations in whole fish averaged 80 ng/g for lake trout, 62 ng/g for rainbow trout, and 36 ng/g for coho salmon. The levels of HCB increased with body weight for all species. Differences in HCB concentrations among species appeared to be related, in part, to fat content of the fish. The mean HCB concentrations in fat were estimated to be 0.5 μg/g for lake trout, 0.7 μg/g for rainbow trout, and 0.5 μg/g for coho salmon.

Environmental samples from a lake subjected to fish kills caused by spills of pentachlorophenol (PCP)-containing wood treating wastes showed that concentrations of PCP and PCP-degradation products in water, sediment, and fish remained high for several months following each kill.[278] Bluegills (*Lepomis macrochirus*) exposed to sublethal PCP concentrations in the laboratory rapidly accumulated PCP (with the highest concentration in the bile, followed by liver, gills, and muscle) but eliminated most of the PCP within 16 days when placed in clean water.

Japanese medaka (*Oryzias latipes*) embryos exposed for 24 and 48 hours to the water soluble fraction (WSF) of crude oil (155 ml WSF/l) had significantly shorter incubation times than control embryos and showed increased opercular movement.[279]

The early hatching medaka appeared smaller than control larvae. Sprague and Logan [280] investigated the separate and joint toxicity of substances used in drilling fluids for oil exploration to rainbow trout. Of the 21 tested materials, paraformaldehyde, capryl alcohol, and five surfactants were the most toxic, with LC_{50}s of less than 100 mg/l. Least toxic were bentonite, barite, and several organics. Three used drilling fluid samples gave LC_{50}s of 0.83–5.3% (Table I). No consistent picture for joint action in mixtures was observed. Additive action, less-than-additive action, and antagonism were all seen in various simulated fluids or mixtures.

Moles *et al.*[281] determined the sensitivities of various species and life stages of Alaskan freshwater and anadromous fishes to benzene and to the water soluble fraction of Prudhoe Bay crude oil. In 96-hour static toxicity tests, the salmonids were consistently the most sensitive species, and species differences between juvenile salmonids were small (Table I). Of the early life stages, emergent salmon fry were the most sensitive. Out-migrant salmonids tested in seawater were two to three times more sensitive than out-migrants tested in freshwater.

The acute toxicity to goldfish (*Carassius auratus*) of many petroleum-derived chemicals was determined by Bridie *et al.*[282] (Table I). Overall, the glycols and glycol ethers were the least toxic (most LC_{50}s >5 000 mg/l), while the carboxylic acids, epoxy compounds, halogenated hydrocarbons, hydrocarbons, and other industrial chemicals were the most toxic (most LC_{50}s <500 mg/l). The alcohols, amines, and ketones varied considerably in toxicity within each group (LC_{50}s 1 to >5 000 mg/l). The amines and carboxylic acids were less toxic at pH 7.0 than at their normal basic or acidic pH, respectively.

Orr *et al.*[283] determined the toxicity of kerosene to fathead minnows and found kerosene to be more toxic in a static 24-hour test (LC_{50} 5 000 µl/l) than in 96-hour flow-through tests (LC_{50} 8 980 µl/l).

Calamari *et al.*[284] proposed an assessment of the toxic effects of chemicals on aquatic life, using a series of laboratory tests on organisms from three different trophic levels then applied this scheme to dimethylamine. Comparing the results of tests on algal growth inhibition, acute toxicity, and reproduction of the crustacean *Daphnia magna* and acute toxicity and embryo-larval survival and growth of rainbow trout, the authors found embryo-larval survival of trout to be the most sensitive effect measured (Table I). Water hardness was selected as the environmental parameter most likely to affect toxicity.

Maki [285] compared the sensitivity of *Daphnia magna* 3-week chronic tests with 1-year fathead minnow chronic tests. He found a high correlation for six surfactants and one detergent builder and a significant but lesser one for metals, PCB isomers, and pesticides. *Daphnia* life cycle tests were suggested as a useful substitute for longer fish chronic tests. The toxicity of alkyl ethoxylate to fathead minnows was measured by Maki *et al.*[286] In tap water the 96-hour LC_{50} was about 1.2 mg/l, and in secondary effluent from a municipal treatment plant the 96-hour LC_{50} was about 2.5 mg/l. Toxicity disappeared within 24 hours because of decomposition, and breakdown products were not toxic in the amounts present.

Birge *et al.*[287] conducted static-renewal and continuous-flow embryo-larval bioassays of four PCBs and three proposed PCB-replacement compounds with five fish species and calculated LC_{50} values at 4 days posthatch (Table I). Studying the relationship between the toxicity and accumulation in goldfish of seven chlorophenols, Kobayashi *et al.*[288] found that toxicity and bioaccumulation ratios increased as the number of chlorine atoms increased (Table I). The 24-hour LC_{50} and concentration ratio for phenol (no Cl) were 0.27 mg/l and 475; for PCP (5 Cl) they were 60 mg/l and 1.9. It appeared that an increase in the number of chlorine atoms promoted accumu-

lation and led to concentration of certain lethal levels (roughly 100–200 μg/g body weight).

Chapman and Shumway [289] studied the effects of sodium pentachlorphenate (Na-PCP) on the early developmental stages of steelhead trout (*Salmo gairdneri*) and found that continuous exposure from fertilization to complete yolk absorption produced mortality at lower Na-PCP concentrations than exposure to Na-PCP during portions of this period. With continuous exposure, 100% mortality occurred at 40 μg Na-PCP/l, but there was little mortality at 10 or 20 μg/l. However, exposure to Na-PCP in water of lowered, though generally nonlethal, O_2 levels (3 and 5 mg O_2/l) resulted in complete mortality at 10 and 20 μg Na-PCP/l, respectively. Exposure to Na-PCP disrupted energy metabolism, as shown by decreased alevin dry weight at hatch, delayed hatch, reduced yolk utilization efficiency and growth, and increased O_2 consumption of alevins.

Studying the effects of Na-PCP on behavior and bioenergetics, Nagendran and Shakuntala [290] observed significant increases in opercular/surfacing activity in the cyprinid forage fish *Puntius ticto* exposed to sublethal concentrations of 1.44–12.96 μg/l. A concentration of 14.40 μg/l was lethal. Fish exposed to all sublethal concentrations of Na-PCP exhibited marked inhibition in daily feeding rates when fed an *ad libitum* diet of *Tubifex tubifex* worms for 25 days. Daily growth rates were 30–50% less than those in freshwater, and Na-PCP lowered food conversion efficiency from 29.59% in freshwater to 19.20–25.06% in Na-PCP exposed fish.

Iwama and Greer [291] reported a 96-hour LC_{50} of Na-PCP to juvenile chinook salmon (*Oncorhynchus tschawytscha*) in soft water (Table I). The continuous-flow bioassay was conducted using a high loading density (13.8 g fish per litre). Intermittent-flow exposures of larval and early juvenile fathead minnows to hexachlorocyclopentadiene, hexachloronorbornadiene, and heptachloronorbornene by Spehar *et al.* [292] resulted in 96-hour LC_{50} and 30-day LC_{50} values of 7.0 and 6.7 μg/l, 188 and 123 μg/l, and 85.6 and 60.1 μg/l, respectively. Bioconcentration factors were uniformly low for hexachlorocyclopentadiene but were higher and proportional to exposure concentration for the other compounds tested (Table I).

Alexander and Clarke [293] reported that phenol was better than sodium azide, Na-PCP, $CuSO_4$, and dodecylsodium sulfate in detecting differences in sensitivity among groups of rainbow trout. Phenol was used to detect differences in sensitivity among strains of trout and made is possible to discern the effects of starvation, temperature stress, and pre-exposure to 0.04 mg Cl_2/l but not the effects of brands of food or high mortality during holding. For the various groups of trout, 48-hour LC_{50} values ranged from 7.7 to 10.4 mg phenol per litre (Table I). The authors discussed the limitations of phenol or any chemical as a reference toxicant to identify groups of fish unsuitable for bioassays.

Bohm *et al.* [294] reported 48-hour LC_{50}s for the electroconductive resin ECR 77, used in the production of electrophotographic paper, for carp, rainbow trout, and guppies (*Poecilia reticulata*) (Table I). Kaolin added to the resin at 1:4 reduced toxicity by three to seven times. The acute aquatic toxicity of the irritant compounds known as "CS gas" (orthochlorobenzylidene malononitrile) and its two principal hydrolysis products was studied by Abram and Wilson. [295] The authors found them toxic to rainbow trout at low mg/l concentrations and suggested that an equimolar mixture of the hydrolysis products behaved synergistically (Table I).

Reduced fertilization in rainbow trout was seen after exposure of gametes to two fatty alcohols. [296] Hexadecanol induced a significant drop in fertilizing ability of sperm after 20- and 40-minute exposures to 1 mg/l and 1 g/l. Octodecanol reduced sperm fertilizing ability only after exposure to 1 g/l. Significant reductions in fertility occurred following 40-minute exposure of eggs at 100 mg octodecanol/l but not at 1 g hexadecanol/l. Following simultaneous exposure of egg and sperm, reduced fertilization occurred only for the highest dose of hexadecanol (1 g/l).

The acute toxicity of some solvent extraction chemicals to green algae; to two cellulose-degrading bacteria; and to a fish, rainbow trout, was determined by Dave *et al.* [297] For most aliphatic amines studied (Primene JM-T, Amberlite LA-1, Adogen 283, Alamine 336, and Aliquat 336), inhibition of algal or bacterial growth (EC_{50}s) and mortality of trout (LC_{50}s) were below 10 mg/l. Other chemicals tested (Adogen 383, TBP, HDEHP, NA SUL AS-50, LIX 64N, Versatic 10, and 2-ethylhexanol) were less toxic, having EC_{50}s and LC_{50}s between 3 and 1 000 mg/l (Table I).

Narbonne [298] reported that accumulation of PCB by grey mullets (*Chelon labrosus*) fed a French PCB (Phenoclor DP6) for 30 days was not age dependent but was probably dependent on food intake. After 30 days of feeding 50 μg PCB/g, a whole body concentration of 5.1 μg/g was reported for 2.5-year-old mullets and 7.4 μg/g for 3.5-year-old mullets.

Guiney *et al.*[299] exposed sexually immature rainbow trout to the PCB isomer 2,5,2′,5′-tetrachloro [^{14}C]biphenyl (4-CB), transferred them to spring water, then followed the distribution and elimination of 4-CB radioactivity for 52 weeks. Egg and sperm maturation and spawning enhanced whole-body elimination of the PCB from the trout. The elimination rate in both sexes increased rapidly during the spawning period. The half-life decreased from 1.76 to 0.52 years in females and from 1.43 to 0.54 years in males because of the redistribution of 4-CB from fatty tissues to the maturing eggs and sperm. This was followed by voiding of these 4-CB-containing gametes during spawning.

Veith *et al.*[300] determined the bioconcentration factors (BCF) of 30 organic chemicals in fathead minnows intermittently dosed with nonlethal concentrations in a flow-through system and found a linear correlation between the log BCF and the n-octanol/water partition coefficient (as log P). BCFs ranged from 2.7 for *tris* (2,3-dibromo-propyl) phosphate to 194 000 for Aroclor 1260 (Table I). Residues of brominated compounds in fathead minnows exposed to brominated wastewater effluent were reported by Kuehl *et al.*[301] They reported 15 bromine compounds and bioconcentration factors of <10–1 300 times for three selected compounds of bromine.

Uptake and distribution of ^{14}C-PCB in juvenile Atlantic salmon exposed to ^{14}C-2,2′,4,5,5′-pentachlorobiphenyl in either fresh water or sea water was compared by Tulp *et al.*[302] The ^{14}C-PCB concentration in whole body was two to three times higher in freshwater-exposed fish than in seawater-exposed fish; at 96-hours, ^{14}C-PCB concentrations were about 2 000 and 750 μg/kg, respectively.

Broyles and Noveck [303] found that the route of entry, the tissue concentration, and the distribution of ^{14}C-labeled 2,5,2′,5′-tetrachlorobiphenyl (TCB) in lake trout depended on the developmental stage at which exposure occurred. Analysis of eggs exposed for 4 hours to water containing 500 μg TCB/l showed that the TCB passed through the egg chorion and accumulated in the yolk. Data resulting from water exposures to sac fry and fry after yolk absorption showed a more rapid and higher accumulation of TCB, achieving 6μg TCB/g in 4 hours with 70% of the ^{14}C-label in fry tissues other than the yolk. Broyles and Noveck [304] also exposed lake trout and chinook salmon sac fry to ^{14}C-labeled 2,4,5,2′,4′,5′-hexachlorobiphenyl (HCB) in a static system for 17 days. The chinook fry accumulated HCB 450-fold to 3.6 μg HCB/g, and the lake trout 950-fold to 7.6 μg HCB/g. Subsequently, all HCB-treated fry died. The amount of ^{14}C-HCB in the dead fry was constant and independent of time of death.

The accumulation and elimination of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), α-hexachlorocyclohexane (α-HCH, β-HCH, γ-HCH), and 2,4,6,2′,4′,6′-hexachlorobiphenyl (2,4,6,2′,4′,6′-HCB) in carp was determined by Sugiura *et al.*[305] The 2,4,5-T was accumulated only in the gall bladder, but α-HCH, β-HCH, γ-HCH, and 2,4,6,2′,4′,6′-HCB were commonly accumulated in the adipose tissue in the skull, the intraperitoneal adipose, the adipose around the spine, and the gall bladder. Excretion was nearly complete for 2,4,5-T and γ-HCH within 2 days, but α-HCH and β-HCH remained in the adipose in the skull and the gall bladder in lower concentrations, while 2,4,6,2′,4′,6′-HCB remained in almost the same amount as accumulated. The results showed that elimination becomes slower according to the increase in the n-octanol/H_2O partition coefficient.

Another study determined the accumulation of α-HCH (α-hexa-chlorocyclohexane), β-HCH, γ-HCH, 4-chlorobiphenyl, 2,3,4,5-tetrachlorobiphenyl, and 3,4,3′,4′-tetrachlorobiphenyl in carp, brown trout, golden orfe (*Leuciscus idus melanotus*), and guppy and demonstrated that the relative accumulation factors of compounds at steady-state were practically constant for any fish species tested.[306] Moreover, the accumulation factors of one compound in different fish species were proportional to the fishes' lipid content. The authors felt that the partition coefficient of a compound and the lipid content of a fish were the dominant factors determining accumulation factors and constants in fishes. Equilibrium accumulation factors (=bioconcentration factor) for the test compounds are given in Table I.

Inui *et al.*[307] studied the behavior of ^{14}C-labeled-2,6-di-tert-butyl-4-methylphenol (^{14}C-phenyl-BHT) in a model aquatic ecosystem

for 28 days. When added to either the soil or the water, only 0.5–10% of the radioactivity was translocated to the fish. BHT was rapidly degraded in the aquatic system to numerous polar intermediates, with many formed by fish *in vivo* by metabolism. The bioaccumulation potential of 2,3,7,8-tetra-chlorodibenzo-para-dioxin (TCDD) for mosquitofish (*Gambusia affinis*) and fingerling channel catfish (*Ictalurus punctatus*), as well as for algae, snails, and daphnids, was estimated in another model ecosystem.[308] The various organisms accumulated TCDD to averages of 2 000–7 000 times the water concentrations of 0.05–1 330 ng TCDD/l (Table I). Tissue equilibrium concentrations occurred in 7–15 days. The author observed that the TCDD was acutely toxic to mosquitofish at 3 ng TCDD/l, with all fish dead in 14 days.

As part of a study to assess the fate of 3,3′-dichlorobenzidine (DCB) in aquatic environments, Sikka *et al.*[309] exposed bluegills to 0.5 and 2.0 mg ^{14}C-DCB/l in a static system and studied DCB uptake and metabolism. DCB was bioconcentrated 132–554 times in 96–120 hours, with mortality occurring before establishment of an equilibrium between fish and water. An acid-labile conjugate of DCB was the only metabolite detected.

Melancon *et al.*[310] studied the uptake and elimination kinetics and biotransformation of trichlorobenzene in rainbow trout exposed in static or continuous-flow systems. When evaluated by a computer program, muscle concentrations from an 8-hour exposure at 0.012 mg/l and a 35-day exposure at 0.02 mg/l gave BCFs of approximately 100, while those from 4-day exposures at 0.001 and 0.01 mg/l gave BCFs of about 400 (Table I). Bile from trout exposed for 24 hours to 0.25 mg/l contained 5 μg trichlorobenzene/l and 10 μg/ml of biotransformation products. Muscle and liver contained 0.8 and 3.7% biotransformation products, respectively.

The skin and epidermal mucus was found to play a role in the uptake and release of naphthalene by rainbow trout.[311] Regardless of the mode of exposure (fed, i.p. injected, or water exposed), trout skin accumulated significant concentrations of naphthalene and its metabolic products; however, levels in skin were higher from water exposure. From all exposure routes, initially high naphthalene levels declined more rapidly than metabolite concentrations, suggesting that the epidermal mucus aids in the excretion of the hydrocarbon.

Kuehnhold and Busch[312] found that naphthalene and 3,4-benzopyrene accumulated more rapidly and penetrated the chorion more readily than did n-hexadecane following exposure of Atlantic salmon embryos to low μg/l concentrations of these hydrocarbons. Hydrocarbons that penetrated the chorion were accumulated mostly in the yolk and egg fluid.

Another study found that carp rapidly accumulated ^{14}C-diisopropylnaphthalene (^{14}C-DIPN), a PCB replacement in carbonless paper, from water and reached an equilibrium concentration (BCF = 203) within 2 weeks.[313] After 24 hours of exposure, the radioactivity of ^{14}C-DIPN was found in gall bladder and hepatopancreas, in adipose tissues of the skull, in the visceral cavity, and around fin bases and vertebrae. An elimination study showed that DIPN was metabolized and excreted into the intestine with the bile. The ^{14}C-DIPN half-life was 56 hours.

Darrow and Addison[314] reported that brook trout (*Salvelinus fontinalis*) rapidly accumulated and cleared n-octano-1-^{14}C-hydroxamic acid (OHA), a parent compound of an experimental iron ore flotation agent. Appreciable amounts of the accumulated material were evidently converted to the corresponding carboxylic acid, then further metabolized by way of the lipoclastic cycle.

Comparing the relative accumulation of ^{36}Cl-labeled Bunker C oil by rainbow trout exposed to either Bunker C oil or Bunker C oil emulsified in Oilsperse 43, McKeown and March[315] showed higher tissue accumulation of the oil from the oil/dispersant mixture. ^{36}Cl-radioactivity was significantly higher in the liver and kidney and somewhat higher in muscle after exposure to the emulsifier/oil mixture. Radioactivity in gills was similar for both treatments, suggesting that the dispersant increased movement of the oil across the gill.

Kikuchi *et al.*[316] followed the uptake, distribution, and elimination of labeled surfactants, sodium laurylbenzenesulfonate (^{35}S-C12-LAS), and sodium lauryl sulfate (^{35}S-C12-AS) in carp exposed to 1.1 mg LAS/l or 0.85 mg AS/l. The surfactants were readily absorbed, principally through the gills, and were rapidly distributed throughout the body. High concentrations were found in gills, blood, hepatopancreas, kidney, and gall bladder. Elimination from fish transferred to clean water was relatively rapid in most tissues and organs except for the gall bladder, which still had in-

tensively localized ^{35}S-radioactivity after 48 hours.

Uptake and elimination of ^{35}S-C12-LAS and ^{35}S-C12-AS, both at 0.5 mg/l, was also studied in carp.[317] Equilibrium of LAS was reached in 48 hours, resulting in BCFs of 20, for whole body, 30 for hepatopancreas, and 9 000 for gall bladder. The ^{35}S-C12-AS radioactivity reached steady state in 24 hours in whole body and gall bladder (BCFs of 4 and 50, respectively), while hepatopancreas reached a maximum at 24 hours (BCF = 50), then decreased during the remainder of the 72-hour exposure. The elimination of ^{35}S-C12-LAS was slower than ^{35}S-C12-AS in whole body, hepatopancreas, and gall bladder. More than 50% of ^{35}S-C12-LAS remained in the whole body after 5 days in freshwater, while ^{35}S-C12-AS was reduced by half in 3 days. The authors also reported that the BCF for LAS increased with increased water hardness (0–200 mg as $CaCO_3$/l) and that the average survival time of carp decreased with increased hardness during exposures to 8 and 18 mg LAS/l.

Verma *et al.*[318] analyzed the activities of alkaline and acid phosphatases and succinic dehydrogenase enzymes in the liver and kidney of *Channa punctatus* and *Cirrhina mrigala* after a 15-day exposure to sublethal concentrations of Swascal IP and a 30-day exposure to sublethal concentrations of sodium lauryl sulfate anionic detergent. Alterations noted in the enzyme activities were more pronounced in liver than in kidney and were generally dependent on concentration. Concentrations of 2.8–9.75 mg Swascol IP/l inhibited alkaline phosphatase activities in liver of both species from 12 to 42% acid phosphatase activities from 15 to 37% and succinic dehydrogenase activities from 8 to 23%.

Abel[319] found, in acute exposures of rainbow and brown trout to sodium lauryl sulfate, that there was no consistent relationship between gill pathology and irregularities in the toxicity curves. The mode of action was probably a complex series of events that could not be simply related to gill pathology. The effect of Swascofix RE45 containing ABS on enzymes in *Mystus vittatus* was determined by Verma *et al.*[320] A concentration of 0.4–0.6 mg/l reduced the specific activity of sodium potassium and magnesium ATPase after 15 and 30 days of exposure.

Bolis and Rankin[321] demonstrated concentration-dependent vasodilation in isolated Pacific salmon (*Oncorhynchus* sp.) gills perfused with anionic detergent LAS (linear alkylate sulfonate). LAS, in concentrations of 1–3 mg/l, caused increases in flow very similar to those produced by the vasodilatory hormones, adrenaline and isoprenaline. The effect of LAS was partly blocked by propranalol, a β-adrenergic antagonist, suggesting involvement of β-adrenergic receptors.

Newsome and How[322] measured the 96-hour LC_{50} of a detergent builder, trisodium carboxymethyloxysuccinate, to goldfish and rainbow trout. The respective LC_{50}s were 2 900 and 2 300 mg/l. The builder was absorbed very little by the body.

Brook trout were fed Aroclor 1254 (PCB) or 3-methylcholanthrene (3-MC) over an 18-day or 25-day period, respectively, in a study of the induction of hepatic MFO enzymes.[323] Trout fed PCB retained about 20% of the dose fed, yielding 39 μg PCB/g of fillet. These fish showed a significant increase in ethoxycoumarin O-de-ethylase activity and slight but nonsignificant increases in aniline hydroxlyase activity and in cytochrome P-450 and microsomal protein concentrations. Trout fed nine doses of 0.35 mg 3-MC showed significant increases in ethoxycoumarin O-de-ethylase, amiline hydroxylase, and aldrin epoxidase activities and in cytochrome P-450 and microsomal protein concentrations.

In another study,[324] fish fed Aroclor 1254 over a 15-day period bioconcentrated 50 μg PCB/g in the fillet with no obvious selectivity of any PCB isomer. Microsomal protein and cytochrome P-450 concentrations were significantly higher in Aroclor 1254-fed trout, and O-de-ethylase activity increased 4-fold. Trout fed XFS-4169L (dielectric fluid C4) showed only slight increase over control values in most MFO enzymes; only the cytochrome P-450 concentration was significantly increased and O-de-ethylase was unchanged. Very little XFS-4169L was deposited in the fillets.

Close correlations between morphologic changes in the endoplasmic reticulum (ER) and induction of microsomal mixed-function (MFO) enzymes in channel catfish livers following acute 7-day and sublethal 21-day exposures to Aroclor 1254 (PCB) were shown by Hinton *et al.*[325] and Klaunig *et al.*[326] Activities of cytochrome P-450, cytochrome b_5, and NADPH-cyt-c-reductase increased in both the acute and sublethal exposures. In addition, aminopyrene demethylase activity increased following the sublethal exposure.

Larger increases in MFO enzyme activities characterizing the sublethal PCB response accompanied extensive changes in ER, including increases in rough ER and structural alterations and increases in smooth ER.

Förlin and Lidman [327] studied the influences of pH, temperature, SKF 525A, α-naphthoflavone, testosterone, carbon monoxide, *in vivo* pretreatment with PCBs, and other factors or the *in vitro* O-demethylation of p-nitroanisole by the rainbow trout liver mixed-function oxidase system. Reaction inhibition by carbon monoxide indicated involvement of the microsomal cytochrome P-450. The PCB pretreatment increased the apparent reaction velocity six to 7 times. The PCB-induced 0-demethylation was markedly inhibited by α-naphthoflavone, while SKF 525A and testosterone strongly inhibited both PCB-induced and untreated rainbow liver enzyme activities.

Induction of benzo(a)pyrene monooxygenase (BPMO) in carp given intraperitoneal (i.p.) injections of benzo(a)pyrene, aflatoxin, crude oil, and extracts from contaminated sea water was observed by Kurelec *et al.*[328] Comparing results from *Salmonella* mutagenicity tests (the Ames test), carp tests, and BPMO levels in fish caught in contaminated sea water, the authors felt that bacterial mutagenicity tests combined with measurement of BPMO induction in i.p.-injected carp could be a useful method of detecting mutagenic-carcinogenic xenobiotics in the aquatic environment.

The effects of polyaromatic hydrocarbons (PAH) on the microsomal liver enzymes in rainbow trout were examined by Gerhart and Carlson.[329] In general, the higher molecular weight compounds caused MFO induction. Cytochrome P-450 levels were elevated by i.p. injection with Aroclor 1254, pyrene, chrysene, and benzo(a)pyrene [B(a)P]. Aryl hydrocarbon hydroxylase was induced by Aroclor 1254 and B(a)P, while aniline hydroxylase activity was enhanced by Aroclor 1254, B(a)P, and chrysene. When fish were exposed to a mixture of pyrene and fluoranthene or B(a)P in water, bioaccumulation of B(a)P induced MFO activity but bioaccumulation of pyrene and fluoranthene did not (Table I). Based on their experiments, the authors predicted that tissue concentrations above 300 μg B(a)P/kg would result in MFO induction in rainbow trout.

The ability of several methylenedioxyphenyl compounds and paraoxon, a known potent esterase inhibitor, to inhibit the hydrolysis and oxidation of di-2-ethylhexyl phthalate (DEHP) by *in vitro* rainbow trout tissue preparations was examined by Melancon and Lech.[330] In addition to paraoxon, only the methylenedioxyphenyl compounds with long side chains (piperonyl butoxide and tropital) inhibited the hydrolysis of DEHP by trout liver subcellular fractions and trout serum.

Hashimoto *et al.*[331] studied the metabolism of 2-mercaptobenzothiazole in carp. In starved carp most of the chemical was found in the bile at 72 hours, but if the fish were fed, the chemical was excreted into the water rapidly, probably through the urine.

Hiraoka *et al.*[332] injected carbon tetrachloride (CCl_4) into the dorsal aorta of rainbow trout and evaluated changes in tissue histology and in blood parameters. Necrotic areas in the liver were accompanied by changes in plasma constituents, while hematological values were unchanged. In the plasma, the albumin fraction and lipoproteins decreased whereas certain globulin components increased. CCl_4 injection decreased plasma lipid levels, mostly because of a decrease in triglycerides. An intraperitoneal injection of rainbow trout with 0.25–2.0 μg CCl_4/g caused significant reduction in total plasma protein concentration at 12, 24, and 26 hours posttreatment.[333] At 24 hours posttreatment, plasma albumin concentration was reduced only in fish given 2.0 μg CCl_4/g.

A similar study reported that plasma GPT activity is a sensitive indicator of CCl_4 toxicity in rainbow trout.[334] The author felt that plasma enzyme measurements are useful to assess chemically-induced organ damage in fish. Darrow *et al.*[335] found that brook trout exposed to 3 mg decanohydroxamic acid/l, a concentration lethal after 7 hours, showed classical symptoms of hypoxemia, including swelling of erythrocytes, increased serum osmolality, and increased lactate concentrations, but no increase in erythrocyte counts. These symptoms of respiratory interference could not be attributed to changes in surface tension of toxicant solution affecting gas exchange at the gills, nor to direct interactions between the toxicant and hemoglobin.

Groch and Svobodova [336] reported pronounced histological changes in carp, but not in rainbow trout, after intoxication with Slovafol 905, 900, and 915, ethoxyl-substituted isononylphenols. Congestion was observed in gill fringes and filaments, accompanied by atrophy of respiratory epithelium

and circulatory disturbances in most tissues and organs examined.

The *in vitro* toxicity of eight known mutagens or promutagens on three fish cell lines and a human foreskin fibroblast cell culture was studied by Kocan *et al.*[337] Two distinct responses were observed. In one, increasing numbers of cells died with increasing chemical concentration. In the other, the cells survived but did not replicate. In general the fish cells responded similarly to human fibroblasts. However, bluegill fry (BF2) cells appeared to be more susceptible to the toxic effects of seven of the eight agents tested.

Sister-chromatid exchange (SCE) studies by Kligerman [338] using the central mudminnow (*Umbra limi*) demonstrated its usefulness as an *in vivo* model system for the detection of genetically active substances (mutagens and/or carcinogens). Following i.p. injections of microgram quantities of methyl methanesulfonate (MMS), a direct acting mutagen, or cyclophosphamide (CP), a promutagen, large linear dose-dependent increases in SCE were seen in all tissues examined. Water exposure of mudminnows to neutral red dye (NR) at concentrations of 0.01–12.5 mg/l revealed accumulation of NR in gills and kidneys and significantly increased SCE rates at all concentrations. However, the concentration of dye in the tissues did not correlate with the SCE rate.

INDUSTRIAL AND MUNICIPAL EFFLUENTS

Chlorine and wastewater. The avoidance of golden shiners (*Notemigonus crysoleucas*) to free and combined residual chlorine and to hypochlorous acid (HOCl) was measured by Larrick *et al.*[339] The authors found avoidance to HOCl consistent at a concentration of 0.015–0.017 mg/l and that the average time of each entry was a better index of avoidance than the number of entries. Hypochlorous acid was the determinant species of chlorine avoidance. A similar study described the response of fathead minnows (*Pimephales promelas*) to steep gradients of hypochlorite.[340] The TRC concentration of 0.115 mg/l was the avoidance threshold. In addition, the authors discussed many aspects of avoidance testing based on literature references. Bogardus *et al.*[341] tested the avoidance of monochloramine in five fish species. Avoidance was found to be concentration-related, but behavior, such as schooling, complicated the avoidance response. The avoidance of common shiners (*Notropis cornutus*) to chlorine was measured by Cherry *et al.*[342] The avoidance threshold for TRC was 0.1–0.2 mg/. Variation of avoidance concentration was lowest for HOCl. Chloramine avoidance thresholds were higher, from 0.2 to 0.4 mg/l, depending on acclimation and test temperature.

Fandrei and Collins [343] used 30-minute shock tests to measure the sensitivity of emerald shiners (*Notropis antherinoides*) to total residual chlorine. The fish were about three times more sensitive at 25°C than at 10°C, but age or size had only a minor affect on sensitivity. Zeitoun [344] gave rainbow trout (*Salmo gairdneri*) shock treatments of chlorine at temperatures of 13.5°–26.0°C and concentrations ranging from 1.02 to 3.5 mg/l. A 48-hour recovery period followed. Survival varied with the particular combinations of temperature and chlorine concentration. All blood measurements returned to normal in surviving fish within 24 hours except for plasma hemoglobin, which took 48 hours to return to normal.

The sensitivity of the emerald shiner, common shiner, spotfin shiner (*Notropis spilopterus*), bluegill (*Lepomis macrochirus*), and carp (*Cyprinus carpio*) to five successive 40-minute exposures of monochloramine at three temperatures was evaluated by Brooks and Seegert.[345] Chloramine was more toxic at higher temperatures. Emerald shiners were the most sensitive, and carp and bluegills were the most resistant. Seegert *et al.*[346] included data on chlorine toxicity for several species of fish. The paper contained data on the effects of intermittent chlorine exposure on five riverine species. The authors suggested that 50% of the 30-minute LC_{50} was a safe exposure if exposure was limited to 30 minutes.

Trabalka and Burch [347] tested the toxicity of three chloro-organic synthetic mixtures to carp embryos. Substantially higher concentrations were required to produce lethality than would be expected to occur in natural waters. Chlorophenol was found to contribute most of the toxicity. Largemouth bass (*Micropterus salmoides*) were exposed to spike and square wave exposure patterns of total residual chlorine.[348] It was found that the area under the time-concentration exposure curve was the best way to express the results and that TRC was more toxic at higher temperatures.

Heath [349] evaluated intermittent exposures of free chlorine and chloramine to rainbow

trout, coho salmon (*Oncorhynchus kisutch*), golden shiner, bluegill, and channel catfish (*Ictalurus punctatus*). A major finding was a 3.3- to 4.5-fold difference in TRC LC_{50} values between free chlorine and monochloramine for trout, coho salmon, and golden shiners. Temperature affected toxicity in a minor way. Zebrafish (*Brachydanio rerio*) embryos were successively exposed to a declining concentration of chlorine over a 6-hour period at three stages of development, hatching, and as larvae.[350] Unstressed embryos were unaffected by such exposures declining from approximately 1 mg/l, but hatching was reduced in those subjected to a mild ionic stress from $CaCl_2$ and $MgCl_2$. Larvae were much more sensitive.

Ward and DeGraeve[351] measured the chronic toxicity of a mixed municipal/industrial waste, nondisinfected and disinfected with chlorine, with and without SO_2 dechlorination, ozonated and brominated, to fathead minnows. Dechlorination removed the lethal effects of chlorine, and no lethality was found in brominated or ozonated effluents. No negative effects on growth or reproduction were discernible.

Liden *et al.*[352] exposed goldfish (*Carassius auratus*) to a mixture of six heavy metals at concentrations equal to the Tennessee standards and then to concentrations of chlorine. While no effect was found during the 30-day exposure, the metal-exposed fish were more susceptible to chlorine toxicity than unexposed fish.

Preston[353] described a biomonitoring program designed to determine the toxicity of industrial effluents discharged under the EPA-NPDES program. Mobile equipment was described, and some results from the tests performed on 11 discharges revealed acute toxicity in nine. The acute toxicity of 12 industrial chemicals to freshwater and saltwater organisms was determined by Curtis *et al.*[354] Both organic and inorganic compounds were studied, using fathead minnows. The 96-hour LC_{50} values ranged from 0.15 mg/l for mercuric thiocyanate to 363.8 mg/l for ammonium fluoride.

The effects of the effluent from a large fish farm on the receiving river was studied by Bergheim and Selmer-Olsen.[355] Eutrophication effects were found to be the most significant change, and there was no alteration in the fecal coliform count.

Martin[356] studied the effects of a wastewater lagoon discharge on a receiving stream in Ohio. Some lowering of DO and elevation in N, P, and Cl^- concentration occurred. The changes did not violate standards, and a healthy community of fish and other organisms was found downstream. White and Williams[357] found that chubs (*Leuciscus cephalus*) and perch (*Perca fluviatilis*) grew faster and roach (*Rutilus rutilus*) grew as fast in tertiary treatment lagoons as in the best European populations. Breeding success was irregular, which may have been because of large algal blooms adversely affecting the eggs and young.

The effect of exposure of *Ophiocephalus punctatus* and *Saccobranchus fossilis* to distillery waste was tested by Verma *et al.*[358] A 2.6–3.1% waste for 10–30 days caused reduced alkaline phosphatase and acid phosphatase activity in the liver and kidney of these fish. Five freshwater first species of India were tested for their sensitivity to untreated textile wastes.[359] The 96-hour LC_{50}s ranged from 2.0 to 3.6% waste. The toxicity of an elemental phosphorus discharge from a munition plant to mosquitofish (*Gambusia affinis*) and bluegills was measured by Pearson *et al.*[360] The 96-hour LC_{50} values were 75 and 29 $\mu g/l$ of P_4, respectively. Badr and El-Dib[361] found that fish chromosomal changes were more pronounced in areas containing higher concentrations of industrial wastes. Both chromosomal abberation and mitotic activity were affected.

The decline in and current water quality of the Rhine River was described by Poels *et al.*[362] Sutdies were cited showing that Rhine River water is chronically toxic to rainbow trout and that there was a 20% increase in chromosome breakages in eastern mudminnows (*Umbra pygmeae*) living in the water. Some of the substances responsible for the toxicity effects include various chlorinated hydrocarbons, polycylic aromatics, and such metals as Cd and Hg.

Oxygen consumption of *Labeo rohita* exposed to concentrations of 5–60% waste from five industrial sources was measured by Hingorani *et al.*[363] In every case the O_2 consumption decreased with increasing waste concentration. Bergheim *et al.*[364] studied the effects of the release of silage juice into streams in Norway. The authors concluded that the reduced O_2 caused most of the effects observed in the streams. The effect of aging, sunlight,

dilution water, and sediment on the toxicity of refinery effluent to several fish species was evaluated by Scheir *et al.*[365] The results showed that all were important and pointed out the need for on-site testing instead of central laboratory tests. Steele and Courtois[366] found that acute exposure of rainbow trout to 15% v/v steam condensate increased the mean corpuscular hemoglobin concentration in these fish. Increases in ammonia and carbon dioxide and decreases in DO were caused by the condensate.

Pulp and paper. Verma *et al.*[367] performed toxicity tests to determine the effect of pulp and paper production wastes on *Notopterus notopterus*. Increases in temperature and pH increased toxicity and increases in DO decreased toxicity. The acute toxicity of pulp effluents, with and without anthraquinone and borate, to fathead minnows (*Pimephales promelas*) was tested by Zanella *et al.*[368] and Zanella *et al.*[369] The acute toxicity was not changed by anthraquinone and only slightly increased by borate. No acute toxicity was found after treatment.

Several studies determined the effect of treated and untreated bleached kraft pulpmill effluent on coho salmon (*Oncorhynchus kisutch*) and/or rainbow trout (*Salmo gairdneri*).[370-373] Concentrations ranging from 0 to 0.5 of the LC_{50} for untreated waste and up to 1 for treated waste caused no adverse effects on growth, swimming stamina, and albumin levels but caused changes in carbohydrate metabolism. A comparison of four different end points showed decreasing sensitivity in the following order: blood sugar test, temperature tolerance test and LC_{50}, and sealed-jar DO test. The pH accounted for most of the difference between the toxicity of effluents, which were least toxic around pH 9 and comparably toxic in sea water and fresh water at similar pH values.

The toxicity of five hardwood pulp bleaching processes to fish was evaluated by Wong *et al.*[374] Toxicity was relatable to chlorine concentration and chlorinated organics. Gaseous chlorine reduced toxicity as compared to aqueous chlorination. Rogers *et al.*[375] fractionated hardboard plant effluent to determine the most toxic components to sockeye salmon (*Oncorhynchus nerka*). The authors found that the neutral and resin acid fractions to contained the most toxic constituents.

PHYSICAL FACTORS

The Department of the Environment (Canada)[376] identified problems associated with land development within watersheds containing salmon-bearing streams and presented feasible measures to counteract these potentially deleterious effects upon the aquatic environment. It was found that an alteration of natural watershed characteristics by land development can cause biochemical and biological modifications that degrade the aquatic habitat and affect spawning and rearing of salmon populations.

The predicted effects of exotic fish introduction on the Manitoba fisheries was described by Loch *et al.*[377] The proposed Garrison Diversion Unit was a multipurpose water resource project designed to divert Missouri River water into central and eastern North Dakota for irrigation and for municipal and industrial water supplies, with return flows entering Hudson Bay drainages. Because the resultant impacts were so potentially damaging, the Canada-U.S. International Joint Commission concluded that the proposed fish screens and other amelioration measures were not a sufficient guarantee against interbasin transfer of foreign biota.

Platts[378] discussed the problems confronting the fishery biologist assisting in the development of land management strategies for proper livestock grazing practice. The author outlined the aquatic components affected and identified livestock-fishery interactions.

Dietemann and Sanderson[379] reported on the fish species inhabiting the Piedmont Potomac and examined some of the probable contributing factors involved in the decline of the fishery over the last decade. The authors concluded that fish life in this section of the Potomac was still a diverse and fair resident fishery and suggested several measures for the protection and enhancement of this resource. Factors contributing to the fishery decline included overfishing, lack of effective fish passage facilities at water supply intakes, flow reduction caused by increased water supply withdrawal, sediment pollution, and storm water containing contaminants.

Large numbers of fish of several species were observed dead below the Tallowa Dam on the Shoalhaven River, New South Wales, Australia.[380] Mortality occurred during river flow stoppages associated with construction of the dam. It was concluded that the long-term effect of the dam, however, would be

more damaging to fish populations such as the Australian grayling (*Prototroctes maraena*) and bass (*Percalates novaemaculeatus*), since it had effectively stopped their migration.

Otobo [381] observed that in spite of a decline in fish yields in the Middle River Niger, Nigeria, no fish family had disappeared as a result of dam construction. It was suggested that more dams could be built to provide lakes and thereby offer more opportunities for growing fish.

Some aspects of flathead chub (*Hybopsis gracilis*) life history were investigated by Martyn and Schmulbach.[382] The authors attributed the paucity of older, larger specimens to destruction of suitable habitat by dams. Only 84 km of the Missouri River below Gavins Point Dam, the most downstream dam, was found to be natural and free flowing. The watershed and all its tributary streams were affected by changes in vegetative cover or in-channel changes.

Bowmaker *et al.*[383] discussed the effects of such human influences as foreign species introduction and pollution, but with particular reference to man-made lakes, on the distribution and community composition of southern African fishes. Besides a review of the research carried out within the impoundments at Kariba and Cabora Bassa dams, they emphasized the ecological effects these dams have had on the Lower Zambezi River. Preimpoundment data and data from Lake Kainji, Nigeria, were compared by Sagua [384] with data on fish composition and production at Faku, near Kainji Dam. The *Mormyridae* and *Mochocidae* were abundant in the *preimpounded* river but became less important, while the *Centropomidae* and *Bagridae* were the most prominent fish families near the dam. These findings were discussed in the context of future plans to build more dams along the River Niger. Impact studies carried out on hydroelectric dams in Kenya were discussed by Odingo.[385] Fish life was found to be disrupted with the change from a riverine to lacustrine environment, and it was recommended that action be taken to slow down the rapid erosion resulting in high suspended sediment loads and high silting rates affecting the Kamburu Dam.

The effects of impoundment and regulation on the stomach content of fish at the Cow Green Reservoir site (Upper Teasdale) in England were described by Crisp *et al.*[386] Before impoundment, brown trout (*Salmo trutta*) ate chiefly benthic organisms and terrestrial casualties, while older bullheads (*Cottus gobio*) predominantly ate Ephemeroptera and Plecoptera. After impoundment, earthworms and other inundated terrestrial material formed the bulk of the trout diet for at least 3 years, although Chironomidae, *Gammarus*, and zooplankton became important. Chironomidae and *Gammarus* were the main staple for bullhead, while Chironomidae, microcrustacea, and detritus increased in importance among minnows (*Phoxinus phoxinus*) after impoundment.

According to Fitz and Holbrook,[387] saugers were an important member of the sport fishery of the Norris Reservoir, Tennessee, following impoundment in 1936, but have since declined to 1% of the catch. Since 1938 first-year growth of saugers has declined, but growth of older-age classes has increased. Although the distribution of principal fish species in the upper Lot (France) and its tributary, the Truyere, has been modified by disturbance in the river from construction of large reservoirs, Tourenq and Dauba [388] found that, overall, species distribution in the river have remained normal and that downstream the distribution was characteristic of a lowland river with abundant fish. Kuznetsov [389] considered several aspects of natural fish reproduction, with particular emphasis given to analysis of spawning (time, places, substrates) and the distribution and growth of young fish during formation of the Kujbyshev Reservoir in the USSR. According to Mutenia,[390] roach (*Leuciscus idus*) grew faster in Lokka Reservoir, Finnish Lapland, than in the former, natural Lake Sompio. Growth was most rapid in the first 2 years, and roach reaching sexual maturity were older (6–7 years) as well as bigger than those found in more southern lakes.

A study by Matter [391] demonstrated that sedimentation can be a severe and long-term aftereffect on surface mining, even in the absence of acid drainage. Compared to a similar unimpacted reference stream, benthic invertebrate and fish density, biomass, and diversity were consistently lower in the two mining-impacted streams studied. Inorganic sediment in these streams, caused primarily by continued erosion of mined areas and abandoned haul roads, covered the natural rubble substrate, smoothing the bottom, filling in pools, and interfering with the processing of allochthonous organic material.

Swenson [392] measured the influence of turbidity on distribution, feeding, and interrelationships between major fish populations in western Lake Superior. Laboratory and field tests showed that, among other things, red clay turbidity did not directly influence survival of even the most sensitive life stages of fish in Lake Superior.

The effect of capture stress on plasma enzyme activities in rainbow trout (*Salmo gairdneri*) was investigated by Bouck *et al.*[393] Angling, electroshocking, seining, and direct netting produced significant differences of plasma protein concentration, lactate dehydrogenase activity, or leucine amino-naphthylamidase activity. Creatine phosphokinase activity was elevated by electroshocking, and capture struggles elevated hematocrits. Whaley [394] subjected fantail darters (*Etheostoma flabellare*) and bluegill sunfish (*Lepomis macrochirus*) to levels and patterns of pulsating direct current electricity within ranges commonly used during field collection. Although mortality of fish so exposed increased with increased pulse frequency and exposure time, mortalities were negligible among fish exposed for 15 seconds or less, regardless of the electrical characteristics. Burns and Lantz [395] concluded that largemouth bass (*Micropterus salmoides*) can be collected by electroshocking without altering several blood and tissue values. Shocking had no significant effect on hemoglobin, hematocrit, plasma protein, or percentage water content of tissue in fish that had been allowed recovery times of 0, 1, 3, 5.5, and 19 hours.

MISCELLANEOUS

Ahrens *et al.*[396] investigated the toxicity of the flame retardant Fyrol-FR-2 (*Tris* [2,3-dichloropropyl] phosphate) released from wetted polyester sleepwear to fish. When a 38 × 64 cm portion of treated sleepwear was immersed in water, all goldfish (*Carassium auratus*) died within 3 hours. Water analysis revealed 30 mg Fyrol-FR-2/l. No toxic effects were observed in fish exposed for 96 hours to the same fabric after it was laundered once. Using continuous-flow bioassay tests, Blahm [397] demonstrated that commercial fire retardants were toxic to juvenile coho salmon (*Oncorhunchus kisutch*) and rainbow trout (*Salmo gairdneri*) and attributed the mortality to ammonia in the retardants. Increasing the diluent water pH from 7 to 8 increased toxicity (Table I). Increasing the test temperature from 10° to 20°C did not significantly affect the retardants' toxicity.

Meunier and Boivin [398] reported on the effects of fluorescent markers on the growth of trout (*Salmo irdieus*) and carp (*Cyprinus carpio*). Doses of 50 mg/kg of tetracycline chlorhydrate, 40 μg/g of fluorescein, 80 μg/g of alizarine red S, or 30 μg/g of calcein blue required to obtain good labeling of bone tissue were found to be compatible with optimum growth of the fish. However, these compounds did have an effect on skeletal tissue (calciotraumatic lines) and on bone mineralization. Doses of 500 μg/g of tetracycline resulted in high mortality of both species, with greater mortality of carp. In addition, there was a decrease in growth of the surviving fish.

Mong and Berra [399] measured the response of the central mudminnow (*Umbra limi* Kirtland) chromosomes to x-radiation. Doses of 350–990 R produced increased frequency of aberrant cells, an increased number of aberrations per metaphase, and a decrease in the number of countable metaphases per fish. Goby eggs (*Neogobius melanostomus*) were exposed to ^{14}C radiation.[400] Exposures of 10^{-9} curie/l had no effect, but exposure to 10^{-7} caused considerable mortality. At 10^{-5} curie/l, 30% mortality occurred. Blaylock and Frank [401] irradiated mosquitofish (*Gambusia affinis*) with 500–1 500 rads using a Co60 source. The CTM was affected only by 1 500 rads and at Day 15. The authors also found atrophy of hematopoietic tissue in gonads 5 days after radiation from 1 500 rads.

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